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PANCREATITIS IN THE ELDERLY PATIENT*

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INTRODUCTION AND HISTORY

Since Classon in 1842 described several cases of pancreatitis, a large volume of material has been written concerning the pathophysiologic aspects of this disease. Friedrich in 1875 described pathologic changes varying from inflammation and hemorrhage to necrosis and abscess formation. In 1876 Klebs described pancreatic cysts and abscesses, and at that time implied that the hemorrhages noted in these cases might be caused by the action of pancreatic juice. The phenomenon of fat necrosis was described by Balser in 1882, and in 1891 Langerhans induced experimental omental fat necrosis in the dog with the injection of pancreatic juice. Fitz in 1889 reported on 70 cases and defined hemorrhagic pancreatitis and necrosis, suppurative pancreatitis, and chronic pancreatitis.[22, 60, 66, 76]

The clinical picture of pancreatitis is well documented, but specific information concerning the response of the elderly patient is scanty. In an effort to study this problem the hospital records of 89 patients admitted to the Medical College of Virginia Hospitals from January 1947 through June 1956, over 60 years of age, and with the diagnosis of pancreatitis, were reviewed. At least a 2-year follow-up is available in all cases. Several patients had multiple admissions, but they are tabulated as single patients.

CLASSIFICATION

Pancreatitis has been classified in various ways, but for this series Priestley's classification is modified in part by that of Probstein and Blumenthal.[74, 75] The final classification is as follows:

1. Acute pancreatitis
   a. Transient
   b. Secondary
   c. Fulminating
   d. Hemorrhagic (nonfatal)
   e. Terminal
2. Chronic relapsing pancreatitis
   a. With complications
   b. Without complications
3. Chronic pancreatitis
   a. Mild
   b. Advanced

The transient cases are those with an acute pancreatitis that is relatively mild, usually nonfatal, and apparently heals without residual disease. Those cases classified as secondary are associated with another primary disease in which the pancreas is only secondarily involved, as by penetration of the pancreas by a posterior perforating duodenal ulcer. The fulminating cases are those in which the patients have hemorrhagic pancreatitis and die after a rapid, downhill course. Patients in the hemorrhagic pancreatitis group who survive are listed under the acute hemorrhagic classification. Terminal acute pancreatitis includes those patients who die, and in whom an acute area of pancreatic inflammation is found which is not the main cause of death in the patient.

The patients in this series can be divided as shown in table 1. In this series 50.6 per cent of the patients had an acute transient or edematous form, and only 10.1 per cent had the acutely hemorrhagic type. Acute inflammation was

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TABLE 1

Classification of pancreatitis

Type	No. of Patients	Per Cent of Total Patients	Average Age
			yr.
Acute			
Transient	45	50.6	70.1
Secondary	14	15.7	71.5
Fulminating	8	9.0	67.6
Hemorrhagic	1	1.1	64.0
Terminal	1	1.1	80.0
Chronic			
Relapsing	8	9.0	66.9
Mild	9	10.1	64.1
Advanced	3	3.4	65.6
Total			
Acute	69	77.5	70.6
Chronic	20	22.5	65.6
Group	89		69.3

present in 77.5 per cent, and the disease was chronic in 22.5 per cent.

In a report of 121 cases by MacKenzie there were 80.3 per cent acute, 5.0 per cent relapsing, and 14.7 per cent chronic.[60] This included all age groups, and the similarity of these percentages is notable. The ages of all the patients in this age-limited series are very similar, but the 5-year difference between the chronic and acute cases may well be due to basic etiologic variances.

ETIOLOGIC FACTORS

A. Incidence of association with biliary tract disease. Korte in 1899 noted the frequent association of pancreatitis with biliary tract disease and particularly with stones, and Lancereaux in the same year speculated that stones lodged within the ampulla of Vater could cause pancreatitis by obstruction of the main duct. Halsted's fatal case of pancreatitis with a stone impacted at the ampulla of Vater stimulated the work of Opie, who first reported his common channel theory in 1901.[23, 60, 76] It was hypothecated that a blockage of the ampulla of Vater occurred distal to the junction of the biliary and pancreatic ducts. This may be caused by stone, spasm of the ampulla, fibrotic stricture,[17] edema,[26] tumor, duodenal diverticulum,[16] ascarides,[53] polyps,[95] or a mucous plug.[92] In order to explain the cases without common duct stones, Archibald in 1913 renewed the idea of spasm of the sphincter of Oddi, and in 1919 he first cut this muscle in an effort to relieve pancreatitis. Spasm can be caused by duodenal inflammation, acid stimulation, chronic stenosis, inflammation of the sphincter, psychic stimuli, reflex stimulation from the biliary tract, duodenal hyperacidity, narcotics, and various parasympathetic agents.[27, 76, 92] The obstruction of the ampulla is believed to produce pancreatitis by causing a reflux of bile into the pancreatic duct. The incidence of biliary reflux in patients with or without pancreatitis varies from 0 to 100 per cent, according to the author, the method of examination, and the criteria used, but averages 25 per cent. Occlusion of the sphincter may also block the pancreatic duct and prevent biliary reflux. Bile entering the duct at normal pressures does not cause pancreatitis and has been found within the normal pancreas.[16, 22, 23, 25, 49, 60, 63, 72, 76, 92]

Closely related to the common channel and biliary reflux theory is the relationship of gall bladder disease to pancreatitis, and *vice versa.* Stones impacted within the ampulla of Vater and causing obstruction have been noted to occur in from none to 8.5 per cent of reported cases.[2, 49, 53, 75] Cholecystitis, both acute and chronic, is more frequently found, varying from 25 to 70 per cent of the reported cases. The presence of associated biliary stones varies from 15 to 48.8 per cent of those with cholecystitis.[23, 27, 40, 64, 76]

In the present series 31.9 per cent of the acute cases had associated biliary tract disease, and 65.3 per cent of these had cholelithiasis. The incidence of cholelithiasis for the total acute cases is 21.3 per cent, and, with 4 common duct stones, the total incidence of these is 5.8 per cent (table 2). In the chronic cases biliary tract disease was present at some period in 45.0 per cent, and 62.6 per cent of these had cholelithiasis for an absolute incidence of 25.0 per cent for the chronic cases. The total number of common duct stones was 1, or 5.0 per cent, in the chronic cases.

The total incidence of 33.7 per cent biliary disease for the entire group is within the range reported in the literature, and the incidence of cholelithiasis in these is 64.6 per cent, so that 22.4 per cent of the total group had stones at some time or other. This incidence of associated cholelithiasis is higher than reported in the litera-

TABLE 2

Incidence of cholecystitis and cholelithiasis

Classification	No. of Patients	With Cholecystitis	Total Patients	Cholelithiasis			
				Present		Previous	
			%	*no.*	%	*no.*	%
Acute pancreatitis							
Transient							
Present admission	45	11	24.4	6	55.5		
Previous admission		4	8.9	3	75.0	1	25.0
Total		15	33.3	9	60.0		
Secondary							
Present admission	14	1	7.1	1			
Previous admission		1	7.1			1	100.0
Total		2	14.2	1	50.0		
Fulminating							
Present admission	8	3	26.6	3	100.0		
Previous admission		1	12.5				
Total		3	26.6	3	100.0		
Hemorrhagic	1	1		1			
Terminal	1	1		1			
Chronic pancreatitis							
Relapsing							
Present admission	8	4	50.0	4	100.0		
Previous admission		2	25.0				
Total		6	75.0	4	66.7		
Mild							
Present admission	9	1	11.1	1	100.0	1	100.0
Previous admission		2	22.2			1	50.0
Total		2	22.2	2	50.0		
Advanced	3	None					
Total acute pancreatitis							
Present admission	69	17	24.6	12	70.7	1	5.9
Previous admission		6	8.7	3	50.0	3	50.0
Total		22	31.9	15	65.3		
Total chronic pancreatitis							
Present admission	20	5	25.0	5	100.0		
Previous admission		4	20.0				
Total		8	45.0	5	62.6		
Total	89	30	33.7	20	64.6	4	13.3

ture. The total incidence of common duct stones is 7.3 per cent for this admission. Two others had previous ductal stones removed.

The incidence of surgery in patients with biliary tract disease and pancreatitis is shown in table 3. Cholecystectomies were performed before, during, or after the present admission in 52.2 per cent of the patients with acute, and 87.5 per cent of the patients with chronic pancreatitis who were found to have biliary disease. Exploratory laparotomies were carried out in 34.8 per cent of the acute, and 62.6 per cent of the chronic patients who had biliary disease.

Cholecystectomies were performed in 61.3 per cent of those patients with biliary disease, and the incidence of common duct stones within this group was 25.8 per cent. The incidence of cholecystectomy for the entire group was 21.4 per cent. The mortality for all patients with biliary disease was 19.5 per cent, or approximately the same as for the entire group.

B. Incidence of pancreatic duct obstruction. Rich and Duff first reported that 54.2 per cent

TABLE 3

Incidence of surgery in patients with biliary disease

Classification	No. of Patients	Cholecystectomy				Common Duct Stone		Exploratory Laparotomy		Died	
		Previous	Present	Later	%	No.	%	No.	%	No.	%
Pancreatitis											
Acute	23	5	5	2	52.2	6	26.1	8	34.8	6	25.1
Chronic	8	3	3	1	87.5	2	25.0	5	62.6		
Total	31	8	8	3		8		13		6	
Per cent with biliary disease					61.3		25.8		42.0		19.4

of 24 patients with hemorrhagic pancreatitis had ductal metaplasia, and found an incidence of 18.7 per cent metaplasia in consecutive autopsies of 150 patients.[17, 27] More recent reports show metaplasia in 11 to 66 per cent of normal ducts, however.[53] Experimental ligation of the pancreatic duct induces acute species-specific inflammatory reactions, but all species eventually exhibit atrophy and destruction of the acini.[2, 41, 69] Clinically the coexistent factors of ductal obstruction and active secretion appear of major etiologic importance. Obstruction has been reported due to mucosal edema, tumor, fibrosis, diverticula, parasites,[68] calculi,[27] and duodenal malformations.[46]

Table 4 shows the incidence of pancreatic and actual common duct obstruction in this series. There were 5.6 per cent of the patients who had obstruction, because of stones, on this admission, 2.2 per cent secondary to some type of stricture, 1.1 per cent due to carcinoma, and 1.1 per cent secondary to a penetrating duodenal ulcer. The total incidence is 10.1 per cent.

C. Incidence of alcoholism. Alcoholism is reported to be the second ranking etiologic factor. Friedreich in 1878 made note of the "drunkard's pancreas," and subsequent reports are numerous. The incidence of alcoholic intake varies from 11 to 66 per cent in the clinical reports, and the related incidence of pancreatitis noted at autopsy in patients who died from acute or chronic alcoholism is 42 to 53 per cent. One report of 31 alcoholic patients who died suddenly noted that all had died of acute hemorrhagic pancreatitis. In these patients there was a remarkable absence of associated biliary tract disease.[25, 76]

The method by which alcohol causes pancreatitis is disputed, and it may well be a combination of several factors, *i.e.*, pancreatic damage, duodenitis, duct regurgitation, capillary hemorrhage, or secondary to acid and secretin formation. These patients have an increased incidence of complications, recurrences and pancreaticolithiasis. Methyl alcohol is a very severe pancreatic poison, and 76 to 100 per cent of patients who die after methyl alcohol ingestion have pancreatitis.[25, 27, 76]

TABLE 4

Incidence and cause of common and pancreatic duct obstruction

Classification	No. of Cases	Stone	Obstructed		Duodenal	Total Patients
			Stricture	Carcinoma		
						%
Acute pancreatitis						
Transient	45	2				4.4
Secondary	14			1	1	14.3
Fulminating	8	1				12.5
Hemorrhagic	1					
Terminal	1	1				100.0
Chronic pancreatitis						
Relapsing	8	1	1			25.0
Mild	9		1			11.1
Advanced	3					
Total	89	5	2	1	1	10.1
Per cent total		5.6	2.2	1.1	1.1	

Reference is made to table 5 for the incidence

TABLE 5

Incidence of alcohol intake and biliary disease

Classification	No. of Patients	Alcoholic Intake		Biliary Disease		Died
		no.	%	*no.*	%	*no.*
Acute pancreatitis						
Transient	45	12	26.6	2	16.7	2
Secondary	14	1	7.1			
Fulminating	8	1	12.5			1
Hemorrhagic	1	1		1		
Terminal	1					
Chronic pancreatitis						
Relapsing	8	2	26.0	1	50.0	
Mild	9	2	22.2			
Advanced	3	2	66.7			
Total acute	69	15	21.8	3	20.0	3
Total chronic	20	6	33.3	1	16.7	
Total	89	21	23.6	4	19.1	

of alcoholic intake in this group. Any patient with a history of recent or past alcoholic intake was included, but the veracity of some of these patients is questionable. A history of alcoholic intake was obtained in 21.8 per cent of the acute and 22.3 per cent of the chronic cases, and the associated incidence of biliary disease was 20 and 16.7 per cent, respectively. Caucasians composed 74.1 per cent of the patients, and 25.9 per cent were Negroes. Alcoholic histories were present in 71.4 and 28.6 per cent, respectively, showing no racial preference.

D. Metabolic disorders. Associated with the alcoholic factor is that of metabolic insufficiency or disorder. Alcohol may be implicated within this group also. Aside from the acute effects already noted, chronic dietary insults associated with alcoholism may be an important factor, especially in chronic pancreatitis. Hyperlipemia, methionine antagonists, kwashiorkor, chronic debilitation secondary to other diseases, porphyria, siderosis, and hemochromatosis also are associated with pancreatitis.[27, 49, 60, 67, 76, 92]

In this series pancreatitis occurred in 5 patients with diabetes mellitus: 3 acute transient cases, 1 chronic case, and 1 acute secondary case, for a total incidence of 5.6 per cent. Bossak and Joelson[8] note an incidence of 7.2 per cent in 103 cases of acute adult pancreatitis; 75 per cent of their diabetic patients died.

E. Vascular lesions. Vascular lesions play a very important role, and insufficiency may result from necrosis caused by digestion of the blood vessel wall, venous thrombosis with moist gangrene, arteriosclerotic arterial rupture, purpuric or coagulative defects, or a vasomotor dyscrasia.[58] Arteriosclerosis, endarteritis, atherosclerosis, disseminated lupus erythematosus, and periarteritis nodosa can all cause some degree of thrombosis.[23, 53, 60, 68, 76]

In addition, the age distribution for patients with pancreatitis and cardio- or cerebrovascular disease is essentially the same. Coronary thrombosis and pancreatitis have been correlated with an increased incidence of hypertension, atherosclerosis, cholelithiasis, achlorhydria and diabetes.[49, 75]

Table 6 shows the incidences of hypertension, shock, atherosclerosis, cardio- and cerebrovascular disease. The widely recognized values of systolic blood pressure of 140 mm. of mercury and/or a diastolic blood pressure of 90 mm. of mercury or more were used as the basis for hypertension, with the realization that these figures are considered "normal" for patients in this age group by many physicians. Shock was recorded from the vital signs on admission or while in the hospital. Atherosclerosis was recorded from the clinical and autopsy diagnoses. Electrocardiogram abnormalities, cardiac size, and auscultatory records were used to evaluate the presence or absence of cardiac disease, and the cerebral element was recorded on the basis of clinical evidence.

The incidence of hypertension and shock is essentially the same in both acute and chronic groups, but the incidences of atherosclerosis and cardiac disease was greater in the acute cases. The chronic and acute patients had an incidence of 10.0 and 2.9 per cent cerebral disease, respectively. The relationship between the acute transient type of pancreatitis with hypertension and associated vascular disease is marked.

Experimentally the type of vascular lesion prominent in the Shwartzman reaction has been implicated by Thal and others as a predominant factor in that type of experimental pancreatitis.[68, 79, 87]

Acute allergic reactions have also caused

TABLE 6

Incidence of hypertension and vascular disease

Classification	No. of Patients	Hypertension		Shock		Atherosclerosis		Cardiac		Cerebral	
		no.	%	*no.*	%	*no.*	%	*no.*	%	*no.*	%
Acute pancreatitis											
Transient	45	28	64.6	3	6.8	29	66.0	25	56.8	1	2.3
Secondary	14	11	78.6	3	21.4	5	35.6	6	42.8		
Fulminating	8	6	75.0	3	25.0	5	62.5	5	62.5	1	12.5
Hemorrhagic	1	1									
Terminal	1	1				1		1			
Chronic pancreatitis											
Relapsing	8	6	75.0			4	50.0	5	62.5	1	12.5
Mild	9	6	66.7			2	22.2	3	33.3		
Advanced	3	2	66.7	2	66.7	2	66.7	1	33.3	1	33.3
Total acute	69	47	68.1	9	13.0	40	58.0	37	53.6	2	2.9
Total chronic	20	14	70.0	2	10.0	8	40.0	9	45.0	2	10.0
Total	89	61	68.5	11	12.4	48	53.9	46	51.7	4	4.5

pancreatitis, but none of this series appeared to fall into this category.[77]

F. Infection. Although heavy coliform cultures were obtained in Opie's case, the relationship between infection and pancreatitis is nebulous. No single etiologic agent has ever been isolated from all of the cases, nor has the disease been transmitted from patients to healthy individuals. Pancreatitis may occur as a part of clinical infections with scarlet fever, mumps, typhoid fever, generalized sepsis, diphtheria, infectious mononucleosis and viral hepatitis, and has been transmitted experimentally *via* Coxsackie virus in animals.[27, 76, 90] Positive cultures are noted in 19 to 55 per cent of the peritoneal fluids in patients with acute pancreatitis, however.[58] No relationship to infection was noted in this series.

G. Trauma. Traumatic pancreatitis occurs in some cases, either direct or transmitted. Travers reported the first injury of this type in *Lancet* in 1827. Mortality following pancreatic war wounds has decreased from 80 per cent in the Civil War and World War I to 22 per cent in the Korean War, and at present civilian hospital mortality rates vary from 8 to 15 per cent. Pseudocysts form in 8 to 33 per cent of these cases, and fistulas in about 35 per cent. Electric shock may also cause pancreatitis.[20, 76]

Postoperative pancreatitis is of recent interest. Postoperative amylase elevations occur in 35 per cent of the patients having upper gastrointestinal and biliary surgery. Several factors may be important in causing pancreatitis postoperatively, among them direct operative trauma, duct ligation, vascular insufficiency, anesthetic effects, and tissue damage with proteolytic enzyme release. The mortality rate approaches 66 per cent in these patients.[22, 29, 35, 38, 61, 94, 96]

In this series 1 case of traumatic pancreatitis occurred following an external injury, and this patient survived. Postoperative pancreatitis occurred in 3 cases. One had a cholecystectomy and lysis of adhesions, 1 an excision of a duodenal diverticulum, and 1 a partial excision of the pancreas *via* a Whipple procedure. All had terminal shock resistant to treatment, and all died—2 on the second postoperative day, and 1 on the third.

Various other agents have been implicated, these including the "stress syndrome," psychogenic reactions, neurogenic factors, pregnancy and heredity.[46, 49, 53] None of these factors was obvious in this series.

VITAL STATISTICS

Meyers and co-workers[64] report that the incidence of pancreatitis is approximately 0.1 per cent, and Saint[79] reports it to be 0.22 per cent of all surgical admissions. At the Medical College Hospitals the incidence was 0.062 per cent in 1947, and had risen to 0.294 per cent in 1956. In 1947, 25.0 per cent of the admissions with pancreatitis were above 60 years of age, but this has fallen over the included time span to 13.4

per cent in 1956. In this series, 74.1 per cent of the patients were white, whereas only 58.0 per cent of the hospital admissions were white, showing an increased incidence in this race for this age group.

The disease is more prevalent in the male sex, ranging from 45 to 73 per cent. The average age in clinical series is in the latter part of the fourth decade, but in autopsy series it is in the sixth decade. The percentage of colored patients varies from 17 to 42 per cent, according to the type of institution reporting.[7, 16, 17, 37, 75, 79]

Table 7 shows that the chronic cases of pancreatitis and those occurring secondary to some other disease had the longest hospital stay. The shortest stay was in the fulminating acute cases. The mortality rates are markedly different for the chronic and acute cases. Additionally 62.9 per cent of these admissions occurred in the cold months from November to May.

SYMPTOMATOLOGY

The typical episode of acute pancreatitis is well documented.[52] The pain is thought to be caused by the distention of the pancreatic capsule, retroperitoneal extravasations, chemical peritonitis, obstruction or spasm of the pancreatic or biliary ducts, or duodenal obstruction.[27] Ninety per cent have abdominal pain, and 80 per cent have nausea and vomiting. Hematemesis occurs in approximately 4 per cent. The pain varies, but is in the right upper abdominal quadrant in 20 to 25 per cent, the lower abdomen in 6 to 10 per cent, and epigastric or generalized in the remainder. From 25 to 50 per cent of the patients complain of radiation into the back.[51] Focal involvement can also cause severe pain.[1, 11]

TABLE 7

Correlation of disease type, hospital stay, and mortality

Classification	Total No. of Patients	No. Dead	Percentage of Group Dead	Total Deaths	Hospital Stay
				%	*days*
Acute pancreatitis					
Transient	45	1	2.2	6.7	11.5
Secondary	14	4	28.6	26.7	20.6
Fulminating	8	8	100.0	53.3	5.0
Hemorrhagic	1				24.0
Terminal	1	1	100.0	6.7	9.0
Chronic pancreatitis					
Relapsing	8				12.8
Mild	9				20.6
Advanced	3	1	33.3	6.7	27.0
Total acute	69	14	20.3	93.3	12.7
Total chronic	20	1	5.0	6.7	18.4
Total	89	15	16.9	100.0	14.1

In cases in the present series in which right upper quadrant addominal pain occurred, only 1 had cholecystitis. Eleven patients had no pain on admission. Table 8 shows that 62.8 per cent of the acute transient cases had epigastric pain initially. The over-all incidence of pain was 87.6 per cent, with slightly less than 70 per cent in the epigastrium. Under "Other" are 18 patients with the onset of pain in the lower abdomen, 1 with the onset in the back, and 3 complaining of chest pain initially.

The onset of symptoms immediately following food ingestion occurred in 10.1 per cent of these patients, and 3.4 per cent had previously ingested various amounts of alcohol. In this series 5 per cent of the chronic, and 11.6 per cent of the acute cases followed food ingestion, and 5 per cent of the chronic and 2.9 per cent of the acute cases followed alcoholic ingestion. This is lower than the incidences reported in the literature.

Table 9 shows the incidence of the radiation of pain, with 55.2 per cent of the patients with pain involved. The degree of radiation is more striking in the acute cases; and the incidence of radiation to the shoulder is higher in the chronic cases.

Not shown in any table is that the onset of pain is sudden. In the cases in which it was noted, 34.2 per cent of the total group with pain, it was very similar in both acute and chronic cases. Pain was constant in 93.4 per cent of the chronic patients as opposed to 71.8 per cent of the acute cases. The incidence of constant pain for the entire series was 80.4 per cent.

The duration of symptoms was noted in 74 patients. Of these, 9.5 per cent were admitted within 6 hours of the onset; 25.7 per cent between 6 and 12 hours; 20.2 per cent between 13 and 24 hours; 33.8 per cent between 1 and 7 days;

TABLE 8

Incidence of pain, relation to food and alcohol in immediate preadmission period

Classification	No. of Patients	Intake					Pain			
		Food		Alcohol		Total	Over-all	Epigastric	Right Upper Quadrant	Other
		no.	%	*no.*	%		%	%	%	%
Acute pancreatitis										
Transient	45	7	15.5	2	4.5	44	86.8	72.8	6.8	22.7
Secondary	14	1	7.1			9	64.3	66.7		33.3
Fulminating	8					6	75.0	50.0		50.0
Hemorrhagic	1					1				100.0
Terminal	1					1				100.0
Chronic pancreatitis										
Relapsing	8	1	12.5			6	75.0	83.4		16.6
Mild	9			1	11.1	8	88.0	62.5		37.5
Advanced	3					3	100.0	100.0		
Total acute	69	8	11.6	2	2.9	61	88.4	67.2	4.8	29.5
Total chronic	20	1	5.0	1	5.0	17	85.9	76.6		23.4
Total	89	9	10.1	3	3.4	78	87.6	69.2	3.8	24.7

TABLE 9

Incidence of pain radiation

Classification	No. of Patients with Pain	Radiation		Radiation to:		
				Back	Shoulder	Abdomen
		no.	%		%	
Acute pancreatitis						
Transient	44	36	58.1	42.4	3.8	53.8
Secondary	9	5	55.6			100.0
Fulminating	6	3	50.0			100.0
Hemorrhagic	1	1	100.0			100.0
Terminal	1					100.0
Chronic pancreatitis						
Relapsing	6	5	83.4	40.0	40.0	10.0
Mild	8	2	37.6	33.3	33.3	33.3
Advanced	3					
Total acute	61	35	57.4	31.4	2.8	65.7
Total chronic	17	8	46.1	36.5	3.75	25.0
Total	78	43	55.2	32.6	9.3	53.1

and 10.8 per cent more than a week after the onset of symptoms.

Previous symptomatology is set forth in table 10. Pain, nausea, vomiting and food intolerance were present previously in a significant number of patients. Previous jaundice had been noticed in 13.5 per cent.

The incidence of previous nausea, vomiting and pain was higher in the chronic cases. The incidence of jaundice was essentially the same. The onset of this episode was related to food intolerance in 23.2 per cent of the acute, and 4.5 per cent of the chronic cases. When tabulated in relation to biliary disease, those with biliary

TABLE 10

Correlation of biliary disease and previous symptoms

Classification	No. of Patients	Pain	Nausea	Vomiting	Jaundice	Food Intolerance
		%				
Acute pancreatitis						
Transient						
Biliary disease	15	80.0	53.3	40.0	40.0	20.0
No disease	30	43.4	23.4	16.6	6.7	20.0
Secondary						
Biliary disease	2	100.0	100.0	100.0	50.0	50.0
No disease	12	75.0	50.0	50.0	8.3	41.6
Fulminating						
Biliary disease	3					
No disease	5	60.0	40.0	60.0		20.0
Hemorrhagic						
Biliary disease	1					
Terminal						
Biliary disease	1					
Chronic pancreatitis						
Relapsing						
Biliary disease	6	100.0	83.4	66.6		50.0
No disease	2	100.0	50.0	50.0	50.0	
Mild						
Biliary disease	2	100.0	100.0	100.0	100.0	100.0
No disease	7	42.8	28.6	28.6		42.8
Advanced						
Biliary disease	0					
No disease	3	66.71	33.3	33.3		33.3
Total acute						
Biliary disease	22	63.8	45.5	36.4	38.8	18.2
No disease	47	53.2	31.91	29.8	6.4	25.6
Total chronic						
Biliary disease	8	100.0	82.5	82.5	24.0	62.5
No disease	12	58.4	33.3	33.3		33.3
Total acute	69	5.6	36.2	31.9	14.5	23.2
Total chronic	20	74.0	55.0	55.0	10.0	4.5
Total biliary disease	30	73.4	56.8	50.0	30.0	30.0
Total no disease	59	54.2	32.2	30.5	5.8	27.2
Total	89	60.7	40.4	37.1	13.5	28.1

disease had a higher incidence of pain, nausea and vomiting, and the incidence of jaundice was six times greater. There was little difference in these patients with regard to previous history of food tolerance.

Table 11 shows that 65.2 per cent of these patients had nausea and 58.4 per cent, vomiting at this admission. Associated with the reported incidence of 5 per cent for gastrointestinal hemorrhage at admission is the finding that 6.7 per cent of these patients had bleeding. No attempt was made to correlate admission incidence with or without biliary disease. These figures are similar to those in the literature, with pain being the most prevalent symptom, followed by nausea and vomiting. In this group, 36.2 per cent complained of weakness, mostly in the acute cases. More of the chronic cases had lost weight. These latter two symptoms are recorded here in higher incidence than in the literature.[44]

The incidence of jaundice at the time of admission varies from 10 per cent to 25 per cent.[1, 26, 34, 45] Serum bilirubin determinations were performed on 68.5 per cent of the patients.

TABLE 11

Incidence of nausea, vomiting, hematemesis, weakness, and weight loss

Classification	No. of Patients	Nausea	Vomiting	Gastrointestinal Hemorrhage	Weakness	Weight Loss
		%				
Acute pancreatitis						
Transient	45	75.5	66.7	4.5	35.6	4.4
Secondary	14	52.8	52.8	31.4	42.8	7.2
Fulminating	8	65.0	65.0		50.0	
Hemorrhagic	1	100.0	100.0		100.0	
Terminal	1	100.0	100.0		100.0	
Chronic pancreatitis						
Relapsing	8	50.0	37.6		35.0	
Mild	9	66.7	55.6	1.1	33.3	44.5
Advanced	3				33.3	
Total acute	69	69.6	63.8	7.2	40.6	4.4
Total chronic	20	50.0	40.0	5.0	25.0	24.0
Total	89	65.2	58.4	6.7	37.2	7.9

The measured level of serum bilirubin or the icterus index was used for evaluation. A value for total bilirubin below 1.0 mg. per cent or an icterus index below 6 units was considered normal. Latent jaundice was considered present with levels of serum bilirubin up to 2.0 mg. per cent and the icterus index up to 16 units. Clinical jaundice is shown by levels above these figures. Of the total, 39.4 per cent had no jaundice, 16.3 per cent had latent jaundice, and 44.3 per cent were jaundiced. The incidence of jaundice for the entire group with or without laboratory determinations was 30.4 per cent (table 12).

These values are higher than those reported in the literature. The chronic cases had higher levels of bilirubin than those shown by the acute cases, and there was no difference between those with or without present biliary tract disease.

Other symptoms that may occur but which are not tabulated here are anorexia, chills, fever, disorientation, diarrhea and constipation.[45] In one series 10 per cent of the patients with chronic relapsing pancreatitis were psychotic, and 75 per cent were "nervous." Psychologic testing showed the existence of depression, overconcern with somatic processes, and a high level of hostility with a tendency to project their problems to other people and situations.[56] One patient here was psychotic.

PHYSICAL EXAMINATION

Shock occurred in 12.4 per cent of the patients at some time during the course of their disease, the greater incidence being in the acute secondary and fulminating cases. This is shown in table 6. Death occurred in 28.6 per cent of the secondary and 100 per cent of the fulminating cases (table 7), in comparison to 2.2 per cent of the transient patients dying, giving an incidence of 6.7 per cent for this group. The prognostic value of shock is thus stressed. Shock reportedly occurs in 7 per cent of all cases.[7, 64] The mechanism for its production is not well understood, but may be due *in toto* or in part to hypertrypsinemia, changes in blood coagulability, blood volume changes, or myocardial and/or adrenal changes.[24, 27, 32, 86]

Cutaneous signs occur in up to 10 per cent of the cases. Morbilliform eruptions, the Grey Turner flank or navel discoloration, livedo reticularis, and cyanosis are reported. The latter is the most common and is seen in the acute phase, whereas Turner's sign appears in 4 to 5 days in the hemorrhagic cases only. One of these patients was cyanotic, but this was on the basis of chronic cardiac and pulmonary insufficiency.[1, 57, 83, 91]

The temperature is elevated in 46 per cent of reported cases.[76] In this series the temperature was above 100° F. in 73.5 per cent of the patients, the incidence being slightly greater in the acute

TABLE 12

Incidence of biliary disease and serum bilirubin determinations

Classification	No. of Patients	Determination Done	Bilirubin or Icterus Index		
			Less than 1.0–6	1.1–2.0 to 6–16	Higher
			%	%	%
Acute pancreatitis					
Transient					
Biliary disease	11	10	40.0	20.0	40.0
No disease	34	24	45.8	16.7	37.5
Secondary					
Biliary disease	1	1	100.0		
No disease	13	7	42.8		56.2
Fulminating					
Biliary disease	3	2	100.0		
No disease	5	2		50.0	50.0
Hemorrhagic					
Biliary disease	1	1		100.0	
Terminal					
Biliary disease	1	1			100.0
Chronic pancreatitis					
Relapsing					
Biliary disease	4	4	25.0		65.0
No disease	4	2	50.0		50.0
Mild					
Biliary disease	1	1			100.0
No disease	8	5	20.0	20.0	60.0
Advanced					
No disease	3	1		100.0	
Total acute					
Biliary disease	17	15	56.1	20.0	33.3
No disease	52	33	42.4	16.2	42.4
Total chronic					
Biliary disease	5	5	20.0		80.0
No disease	15	7	27.5	14.3	57.2
Total acute	69	48	43.8	16.6	39.6
Total chronic	20	13	23.1	15.4	61.5
Total biliary disease	22	20	40.0	15.0	45.0
Total number biliary disease	67	41	39.1	17.1	43.9
Totals	89	61	39.4	16.3	44.3

cases. Respirations were above 30 per min. in 28.3 per cent of the series, and tachycardia was present in 56.2 per cent. In the acute fulminating cases tachycardia was present in 93.2 per cent.

The findings on physical examination vary according to the degree of inflammation. Abdominal tenderness occurs in from 62 to 87 per cent, and 34 to 52 per cent of the patients are reported to have true muscle spasm. Hypoperistalsis is present in from 20 to 50 per cent, and the sensorium is disturbed in approximately 50 per cent of the patients dying of this disease.[1, 34, 45, 91] In this series, as shown in table 13, abdominal distention was present in 31.4 per cent, and tenderness was approximately 3 times as common as true spasm. Hypoperistalsis occurred in 23.6 per cent and hyperperistalsis in 9.0 per cent. Of interest is the fact that only 5 of the fulminating cases showed abdominal tenderness, but 4 showed true muscle spasm.

Leukocytosis reportedly occurs in 69 to 79 per cent of cases, being marked in 25 per cent of these. This is less true in acute alcoholics. A hemoglobin value of less than 10 gm. per cent is reported in 23 per cent of cases.[7, 76] Table 14 reveals that this series showed a lower incidence of anemia and of leukocytosis. White cell count elevation was present in only 59.5 per cent. No especial abnormalities in the differential count was noted. Bone marrow was not studied in this series.[82] Eosinophilia may occur with allergic pancreatitis, and lymphopenia has been reported in up to 89 per cent of all cases, the latter being a stereotype response to stress.[43, 50, 59] These latter changes were not noted in this series.

No reports were noted in the literature concerning the presence of hematuria or pyuria, and these were not grossly noted here. Routine urinalysis showed microscopic red cells present in 24.7 per cent, and white cells in 47.5 per cent (table 15). Autopsies were performed on 8 of the 15 patients who died, and 5 of these showed renal pathology. This included nephrosclerosis, tubular degeneration, renal arteriosclerosis, and acute tubular and glomerular degeneration, the latter in 1 patient only. Albuminuria was present in a higher degree in the acute cases. Richman has reported an incidence of albuminuria in 25 per cent of patients. The increased incidence here may well be due to chronic changes secondary to the age of the patient. Richman also reports an incidence of 11.0 per cent glycosuria, similar to the incidence in this group.[76] Note the highest incidence was in the patients with fulminating and chronic relapsing pancreatitis. The incidence of hyperglycemia varies from 5 to 25 per cent of the reported cases.[7, 25, 45, 76] Blood sugar determinations were made in 63 of these patients, and 58.7 per cent of those were elevated. Most of these were mild and transient, and only the diabetics showed a continuing hyperglycemia. All of the fulminating cases had elevated blood sugar levels.

Elevation of the blood urea nitrogen is thought to be a bad prognostic sign. Determinations were performed on 76, and 44.8 per cent were above normal, most patients being transient only.

TABLE 13

Abdominal findings on admission

Classification	No. of Patients	Distended	Tender	Spasm	Peristalsis	
					Hypo-	Hyper-
		%				
Acute pancreatitis						
Transient	45	42.2	91.2	33.3	28.8	8.9
Secondary	14	14.3	78.6	14.6	14.6	
Fulminating	8	50.0	62.5	50.0	25.0	25.0
Hemorrhagic	1	100.0	100.0		100.0	
Terminal	1					100.0
Chronic pancreatitis						
Relapsing	8	12.5	25.0	25.0	12.5	12.5
Mild	9		89.0	11.1	11.1	
Advanced	3	33.3	66.7		33.3	
Total acute	69	37.6	85.6	30.4	26.1	10.1
Total chronic	20	10.0	80.0	15.0	20.0	5.0
Total	89	31.4	84.3	27.0	23.6	9.0

TABLE 14

Hemoglobin and white cell values

Classification	No. of Patients	Per Cent Hemoglobin		Per Cent White Count		
		Less than 10.0 gm. %	Above	Below	10–20,000	Above
Acute pancreatitis						
Transient	45	4.4	89.2	40.0	46.6	15.5
Secondary	14	14.6	7.14	46.4	46.4	14.6
Fulminating	8		100.0	50.0	25.0	25.0
Hemorrhagic	1		100.0		100.0	
Terminal	1		100.0		100.0	
Chronic pancreatitis						
Relapsing	8		100.0	50.0	50.0	
Mild	9		100.0	47.1	33.3	11.1
Advanced	3	33.3	66.7	33.3	66.7	
Total acute	69	5.8	86.0	46.4	45.0	15.9
Total chronic	20	5.0	95.0	45.0	45.0	10.0
Total	89	5.6	88.7	46.2	44.9	14.6

Liver function studies were done in 37 patients and were abnormal in 62.2 per cent. These included the alkaline phosphatase, thymol turbidity, serum cholesterol, cephalin flocculation, and Bromsulphalein retention. In the reported series approximately 25 per cent showed abnormal studies.[36] Usually only 1 or 2 of the several tests performed were abnormal. Note the higher incidence of abnormality in the acute cases as shown in table 16.

Hypokalemia reportedly occurs in 75 per cent of studied cases, probably secondary to body protein breakdown, alkalosis (some instances), nasogastric suction, and intravenous saline infusions, if no replacement therapy is carried out.[25, 76] Electrolyte determinations were performed on 64 patients, and 51.6 per cent of these showed some abnormality. Table 17 shows that 20 patients had abnormalities in the potassium concentration, and in 90 per cent of these there

TABLE 15

Urinalyses

Classification	No. of Patients	Albumin	Sugar	White Blood Cells	Red Blood Cells
		%			
Acute pancreatitis					
Transient	45	64.4	11.1	42.2	31.2
Secondary	14	64.3	7.2	57.2	35.6
Fulminating	8	50.0	25.0	50.0	
Hemorrhagic	1				
Terminal	1				
Chronic pancreatitis					
Relapsing	8	62.5	25.0	50.0	
Mild	9	44.4	11.1	44.4	22.2
Advanced	3	33.3		33.3	
Total acute	69	62.4	11.6	47.8	29.0
Total chronic	20	50.0	15.0	45.0	10.0
Total	89	59.6	12.4	47.9	24.7

TABLE 16

Blood sugar, blood urea nitrogen, and liver function tests

Classification	Blood Sugar Elevated	BUN-NPN* Elevated	Liver Function Test Abnormal
	%		
Acute pancreatitis			
Transient	51.7	24.2	68.2
Secondary	37.5	68.5	83.4
Fulminating	100.0	85.8	
Hemorrhagic		100.0	100.0
Terminal	100.0	100.0	
Chronic pancreatitis			
Relapsing	50.0	20.0	
Mild	75.0	44.4	40.0
Advanced	100.0	40.0	
Total acute	55.2	46.7	50.0
Total chronic	68.8	3.5	28.6
Total	58.7	44.8	62.2

* Blood urea nitrogen-nonprotein nitrogen.

was a hypokalemia. Note that the carbon dioxide combining power changes also.

Serum calcium determinations were low in 84 per cent of the patients in whom they were performed. There were low values in all of the chronic cases. The reported incidence of hypocalcemia is from 23 to 70 per cent. The lowest value found in the literature was 5.0 mg. per cent, with eventual recovery.[7, 37, 44, 76, 93] Hypercalcemia should suggest hyperparathyroidism.[18] There were no elevations of serum calcium values in this series.

Prothrombin levels are shown in table 18. The low values do not include patients on dicoumarol or similar drugs. The serum amylase values are also shown in this table and the incidence of elevation is striking, being low only in the mild chronic cases. The acute cases had more frequent and higher elevations. The amylase values returned to normal within 3.9 days, on the average.

Serum amylase values have received an enormous amount of attention in the recent literature, being elevated in 60 to 91 per cent of the acute, and less in the chronic cases of pancreatitis.[76] Urinary amylase values were not performed. Although they remain elevated somewhat longer, they are dependent upon renal function to a large degree.[77, 80] The serum amylase is relatively nonspecific and may be elevated in many other

TABLE 17

Serum electrolytes

Classification	No. of Patients	Electrolytes		Serum Calcium	
		No. done	% abnormal	No. done	% abnormal
Acute pancreatitis					
Transient	45	34	35.4	14	78.6
Secondary	14	10	90.0	1	7.1
Fulminating	8	8	50.0	3	66.7
Hemorrhagic	1	1	100.0	1	100.0
Terminal	1	1	100.0	1	100.0
Chronic pancreatitis					
Relapsing	8	3	66.7	2	100.0
Mild	9	5	60.0	1	100.0
Advanced	3	2	50.0	2	100.0
Total acute	69	54	40.0	20	80.0
Total chronic	20	10	70.0	5	100.0
Total	89	64	51.6	25.0	84.0

	No. of Abnormalities	% Depressed	% Elevated
Electrolyte			
Sodium	4	75.0	25.0
Potassium	20	90.0	10.0
Chloride	3	33.3	66.7
CO_2 combining power	21	95.4	4.6

TABLE 18

Prothrombin and serum amylase values

Classification	Prothrombin		Serum Amylase		Normal
	No. done	Per cent low	No. done	Per cent elevated	
Acute pancreatitis					
Transient	15	20.0	44	95.5	7.6
Secondary	5	80.0	13	69.2	8.6
Fulminating	1		6	83.4	4.0
Hemorrhagic	1	100.0	1	100.0	2.0
Terminal	1		1	100.0	3.0
Chronic pancreatitis					
Relapsing	3	66.7	6	83.4	5.4
Mild	5		7	28.6	2.5
Advanced			2	100.0	2.0
Total acute	23	34.8	65	89.3	4.5
Total chronic	8	37.5	15	60.0	3.3
Total	31	35.5	80	83.8	3.9

diseases, but this elevation is usually less than 4 to 5 times the normal values.[12, 15, 31, 42, 69, 81]

It has been reported elevated in the following cases: carcinoma of the head of the pancreas, penetrating duodenal ulcer, cholecystitis, renal functional impairment, peritonitis, intestinal obstruction, mumps, salivary gland disease, and the postoperative state. Other instances are: after thyroidectomy, with liver disease, pneumonia, biliary dyskinesia, adrenocortical insufficiency, and after cholangiography.[25, 35, 39, 59, 62, 71, 76] Morphine may cause an elevation, and 2 patients with myocardial infarctions reportedly developed pancreatitis after receiving it.[31, 34]

Stool examinations were not routinely performed for content. Steatorrhea occurred in only 1 of these cases.

Electrocardiographic changes are noted in up to 70 per cent of patients with pancreatitis, and several have been misdiagnosed as myocardial infarctions. The cause of these changes is obscure and speculative.[5, 76] Electrocardiograms were performed on 67.4 per cent of these patients, and 75 per cent were abnormal. There was no type-specific abnormality noted.

Table 19 lists the incidence of positive x-ray findings from a flat x-ray of the abdomen. Some type of small bowel ileus was present in 42.8 per cent, and 21.4 per cent showed some other abnormality consistent with pancreatitis. Calcium in the pancreas was noted in 2 cases. The incidence was higher in acute cases.

Segmental jejunal ileus occurs in 25 per cent of the reported cases.[60, 64] Localized ileus of some type has been reported in up to 85 per cent of these patients, most often being found during the first 12 to 48 hr. after the onset.[4, 7] Other signs include obliteration of the psoas outline, left diaphragmatic elevation, pleural effusion, a colon "cut-off" sign, air levels in the large intestine, and a hazy appearance in the upper midabdomen with a lateral gastric air tilt.[21, 25, 73]

Table 20 shows that 49.4 per cent of the patients had cholecystograms and that 56.8 per cent of these were abnormal, varying from nonfunction to poor function, with or without stones and/or stricture. Nonfunction occurs in approximately 70 per cent of acute pancreatitis cases without cholecystic disease, with function returning later. This is probably caused by a functional failure in water absorption.[51, 76] Forty-one patients had an upper gastrointestinal series performed, and 63.4 per cent were abnormal.

TABLE 19

Electrocardiographic and abdominal x-ray changes

Classification	Electrocardiograms		Abdominal X-rays		% other
	No. done	% abnormal	No. done	% ileus	
Acute pancreatitis					
Transient	30	73.4	15	40.0	20.0
Secondary	11	63.3	3	66.7	
Fulminating	4	65.0	4	50.0	
Hemorrhagic	1	100.0			
Terminal	1	100.0			
Chronic pancreatitis					
Relapsing	6	83.4	3	66.1	33.3
Mild	6	83.4	2		50.0
Advanced	1	100.0	1		100.0
Total acute	47	72.3	22	45.5	13.6
Total chronic	13	84.8	6	33.3	50.0
Total	60	75.0	28	42.8	21.4
Per cent total	67.4	50.6	31.4		

TABLE 20

Cholecystograms and gastrointestinal barium studies

Classification	Cholecystograms		Gastrointestinal Series		% ulcer
	No. done	% abnormal	No. done	% abnormal	
Acute pancreatitis					
Transient	30	51.7	22	50.0	
Secondary	4	65.0	8	65.0	65.0
Fulminating					
Hemorrhagic					
Terminal					
Chronic pancreatitis					
Relapsing	6	50.0	2	100.0	
Mild	3	66.7	8	87.5	12.5
Advanced	1		1		
Total acute	34	56.9	30	56.7	20.0
Total chronic	10	50.0	11	81.1	11.1
Total	44	54.8	41	63.4	27.0
Per cent total	49.4				

There was present some suggestive sign of duodenal or gastric disease with, or secondary to, pancreatic disease. Changes in mucosal patterns, gastric and/or duodenal loop deviation or stasis, and depression of the ligament of Treitz have been described in pancreatitis.[21]

DIAGNOSIS

The list of diseases which may mimic all or part of the pancreatitis picture is long and includes most of the acute intra-abdominal diseases, coronary occlusion, renal colic, and some early pneumonias. Dissecting aortic aneurysms, benign paroxysmal peritonitis, acute porphyria, and lupus erythematosus have been so misdiagnosed.[7, 79, 89, 93] Complications also occur concurrently that may obscure the diagnosis.[13, 14, 65]

TREATMENT

The most important and urgent need of the treatment schedule is the correction of shock by whole blood, plasma, human serum albumin, or dextran. Levophed may be of value when complete vascular collapse occurs.[48, 76] None of this series received cortisone or adrenocorticotrophic hormone although some reports are very encouraging.[10, 33, 78, 85] Others have found these agents to be of no benefit in the treatment of this disease.[47]

Intravenous fluid therapy should be used as necessary. Theoretically, there may be some disadvantage to the use of intravenous glucose, as hyperglycemia is known to stimulate pancreatic secretions experimentally. This is not apparent clinically. Calcium is given as indicated. Hypoglycemia should be avoided.[3, 34, 37, 48] In this series 83.2 per cent of the patients received intravenous fluids, mostly dextrose and saline, but whole blood and plasma were used as indicated.

Antibiotics were given to 88.7 per cent of the patients, and, of those given, the combination of Crysticillin and streptomycin was used in 98.8 per cent. All of the wide range antibiotics except chloramphenicol were used sparingly. These may be useful in preventing secondary suppuration and bronchopneumonia. Theoretically they are best given orally for greater inhibition of intestinal bacteria.[37, 76]

Nasogastric suction was used in 61.8 per cent of the cases, mostly in the acute group. This decreases the degree of distention and the stimulus for increased pancreatic secretion. Some form of antispasmodic or ganglionic blocking agent was used in 67.4 per cent of the cases, with little difference in use according to the type of disease. The nitrites, nitroglycerine, hexamethonium compounds and tetraethyl ammonium compounds have been advocated but were not used in this series.[3, 34, 48, 54]

All of the chronic cases and 72.8 per cent of the acute cases required narcotics for pain relief. Demerol is considered the drug of choice because there is less effect upon the sphincter of Oddi. Intravenous procaine and aminophylline may also be used, but were not in this series. Epidural blocks were used in 6.7 per cent of the cases with uniformly good results when left in continuously. Splanchnic and sympathetic blocks were not used.[34, 38, 48, 54, 58]

Oral feeding was withheld until all distention and ileus had disappeared. Here, on an average, fluids were given orally at 3.3 days, and solid foods, at 6.7 days.

The mortality rates vary with the method of management. Immediate surgery yields rates of

3.2 to 38 per cent, while the nonoperative management yields a rate of 13 to 19 per cent. These have fallen in the last decade from approximately 38 per cent to between 10 and 13 per cent. Mortality in the hemorrhagic form has fallen from 76 to 33 per cent, and in the edematous forms it has decreased from 12 per cent to 5 per cent.[1, 23, 26, 48, 55, 64] Table 21 shows the surgical mortality of this series to be 24.0 per cent, whereas the nonoperative mortality is 14.5 per cent. Table 7 has noted that the total mortality is 16.9 per cent. Of those who died, 2 had postoperative hemorrhagic pancreatitis, and 5 others had some other surgical procedure performed; thus 47.0 per cent of them had had some form of surgery. Table 22 lists the causes of death, and hemorrhagic pancreatitis was responsible for 42.8 per cent of them. If the 2 postoperative cases are added to this, the figure rises to 57.1 per cent.

Of the patients in this series with hemorrhagic pancreatitis, 88 per cent died, but none of acute hemorrhage. However, Aaron[1] reports this is a cause of death and advises exploration if the temperature rises or a mass appears, since these are indicative of suppuration and presaging vessel erosion.

SURGERY

Table 21 reveals that 38.2 per cent of this group had some type of surgical operations, and of these 36.0 per cent had biliary tract surgery, 48 per cent had gastrointestinal surgery, and 4 per cent were explored and/or drained only. The incidence of complications was particularly high and included wound infections, resistant paralytic ileus, pulmonary infections, embolic phenomena, and cardiac disorders. The surgical mortality occurred in the acute cases, although 35.0 per cent of the chronic cases had surgery.

The main indications for surgical exploration in the acute case are either a failure to establish a definitive diagnosis, or biliary tract obstruction. Most authors believe that correction of the biliary disease is the only procedure that should normally be done at this time. Drainage of the lesser sac and the peritoneum does not *per se* drain the pancreas. In 1949, Rhoads, in contradistinction, reported a surgical mortality of 3.2 per cent by exploring all patients with this diagnosis.[33, 78] Most surgical procedures are advocated for the treatment of some form of the chronic disease.

Several operations have been advocated for the relief of pain, but reports are at variance with respect to their success. The relief is usually short lived following sympathectomy, with or without splanchnicectomy, and/or vagotomy, although there are strong proponents of this procedure.[19, 70, 74, 92]

Cholecystostomy, sphincterotomy, and choledochostomy are advocated for the relief of biliary tract pressure. These advocates believe

TABLE 21

Surgery performed

Classification	Surgery %	Biliary %	GI %	Drain, Other %	Complicated %	Died %
Acute pancreatitis						
Transient	8.0	50.0		50.0	75.0	
Secondary	60.0	11.1	89.0		77.8	22.2
Fulminating	50.0	25.0	50.0	25.0	100.0	100.0
Hemorrhagic	100.0	100.0				
Terminal						
Chronic pancreatitis						
Relapsing	37.5	66.7	33.3			
Mild	44.5	75.0	25.0		50.0	
Advanced						
Total acute	26.1	22.2	55.6	16.6	66.7	33.3
Total chronic	35.0	71.4	23.6		23.6	
Total	28.2	36.0	48.0	4.0	56.0	24.0
Nonoperated	71.8					14.5

TABLE 22

Cause of death

Cause	No. Dead	Percentage of All Deaths
Hemorrhagic pancreatitis	6	40
Postoperative pancreatitis	2	14.3
Myocardial infarction	2	14.3
Peritonitis	2	14.3
Carcinomatosis	1	6.7*
Pneumonitis	1	6.7
Hemolytic reaction, blood	1	6.7

* Pancreatitis secondary to tumor emboli.

in the common duct-pancreatic duct reflux theory, or they have shown that the particular patients involved have an element of biliary obstruction. Cholecystectomy is included. If infection or a stone is present, the gall bladder should be removed if possible and the duct explored.[26]

Choledochojejunostomy-en-Y has its advocates as the procedure of choice because this allows complete diversion of the biliary stream from the pancreatic duct.[3, 6, 9] The incidence of cholangitis and stricture is greater with choledochoduodenostomy than with the choledochojejunostomy-en-Y. This is not a procedure to be done immediately, but should follow a cholecystectomy and choledochostomy with choledocholithectomy if necessary. As a clinical trial a period of time should pass before the anastomosis is done, if this should be found necessary.

Sphincterotomy has major advocates, and Doubilet[18] reports that 88 per cent of his surviving patients have been relieved of pain, gained weight, and returned to their occupations. Others do not report the same degree of success, but earlier surgery may give better results.[28, 54] Patients with biliary disease are much more improved than others after any decompressive procedure.

Vagotomy is advocated by some, but the results are equivocal.

Exploration of the pancreatic duct is indicated when calcific pancreatitis is present or there is suspected blockage. The results are good when obstruction is found and removed or later bypassed.[19, 63, 65]

Cysts or abscesses in the pancreas that may arise as complications should be surgically attacked. Marsupialization is not yet entirely abandoned, but most of the procedures advocated are of the internal drainage type.

With the etiologic emphasis shifted to obstruction in the more chronic disease, caudal or distal pancreatectomy with pancreaticojejunostomy of some type may be effective.[30] Subtotal pancreatectomies are also useful where cysts or localized areas of pancreatitis are confined to the body or tail of the pancreas, but where there is more diffuse involvement the relief may be equivocal.[19]

Total pancreatectomy and/or pancreaticoduodenectomy are becoming increasingly better tolerated, and, after all lesser procedures fail, one of these may be indicated. Diabetes is a special problem along with nutritional defects, but these can now be handled fairly adequately.[58]

Ligation of the pancreatic duct results only in atrophy without severe pancreatitis. Reinhoff performed 1 such operation in 1946 and the patient was relieved for the follow-up period of 10 months. Noer[69] reports 2 patients who were relieved by this procedure.

The surgical results vary, and all of the procedures have proponents. The management of these patients with chronic pancreatitis is difficult, and in addition to the attempted therapy with the various surgical procedures, control of the nutritional and other associated psychologic defects is of great importance. In this series none of the patients had advanced pancreatic surgery for their disease.

SUMMARY

A résumé has been presented of the history, classification, and pathologic and pathophysiologic picture of pancreatitis in the elderly patient. The incidence of the various types of pancreatitis were similar in the elderly patients reported in this series to those series including all age groups.

A review of the implicated etiologic factors noted in the surgical literature has been presented and compared with the present series. This series showed a lower percentage of patients with biliary disease, but more of them had associated cholelithiasis. The incidence of common duct stones is the same. The incidence of atherosclerosis and cardiac disease is higher, whereas the incidence of alcoholic ingestion is in the lowest range.

No striking differences were noted between this series of elderly patients and patients of all ages with pancreatitis. These elderly patients tolerate this disease almost as well as younger patients, and the differences in these patients are, in general, those that are exhibited by this age group in most diseases.

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CARDIAC MASSAGE: A REPORT OF 148 CASES*†

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According to Stephenson,[2] the first attempt at cardiac resuscitation was made by Niehaus in 1880, whereas the first successful case was that of Igelsrud in 1901. In the years that followed, sporadic reports of successes and failures with this procedure appeared in the literature. However, it was not until 1947 that more widespread interest was generated by the first report of successful defibrillation.[1] Since then, many reviews have appeared and several medical centers have offered courses in cardiac resuscitation. All of these authorities agree that two critical requirements must be fulfilled within a crucial 3-minute interval from the time of arrest: (1) effective manual systole, and (2) controlled pulmonary ventilation with adequate oxygen.

At Grady Memorial Hospital in the 7 years beginning July 1, 1952, there were 148 cases of cardiac arrest which were treated by immediate thoracotomy and manual systole. Twelve patients survived, a salvage rate of 8 per cent. Although the heart initially responded in 43 per cent, only 26 per cent lived for 24 hours or more. These rates are considerably below the averages found in the literature.

This is a review of these cases, with particular evaluation of the efficacy of attempting cardiac resuscitation outside of the operating area, in cases of trauma, in the extremely ill patient, in cases of hypovolemia, and in the terminal stages of anoxia. Predisposing factors and premonitory signs of cardiac arrest were investigated. Correlations were made of the patients who, although the heart initially responded, died of complications secondary to temporary circulatory failure. A follow-up study was made of the survivors, with reference to present status, future operative risk, and residual deficits of cardiac and neurologic function.

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GENERAL DATA

Location. Eighty-seven arrests occurred in the operating room, whereas 61 took place in other areas of the hospital (table 1). There were no survivors from arrest which occurred outside of the operating room, and the percentages of hearts that responded and of 24-hr. survivals were significantly lower in those other areas. Recently, the recovery room with its maximal nursing coverage has reduced to a minimum the number of arrests that occur during the immediate postoperative period. However, the majority of arrests which occurred outside of the operating room took place in the emergency clinic and were in seriously ill patients or in cases of moderate to severe trauma. Even though there have been no survivors from arrests outside of the operating room, attempts at cardiac resuscitation should be continued in these other areas, provided that there is no specific contraindication.

Age. Although there were more arrests in the fourth decade than in any other, there was a relatively even distribution of approximately 20 cases for each decade up to the eighth, in which group only 7 were found (table 2). All survivors were 37 years of age or older, with the exception of 1 child. One-fourth of all patients in the seventh decade were successfully resuscitated, and these 4 accounted for one-third of all survivors. The oldest survivor, a 75-year-old Negro man, was not only the first case of cardiac arrest successfully massaged at Grady Memorial Hospital; but he was also the oldest in which it was attempted. Thus, age should be no contraindication to energetic resuscitative measures.

Terminally ill. Cardiac massage was useless in the terminal phase of an illness. Out of 15 patients, only 1 lived more than 24 hr.; and he died 40 hr. after massage. These were patients who had died from their disease, and do not represent true cardiac arrest. Even when temporarily resuscitated, death ensued a few hours later.

Groups. All 148 patients were placed in one of three groups (table 3).

TABLE 1

Location	No. of Patients	Responded	Lived 24 hours	Survived
Operating room	87	42	30	12
Clinic	30	7	4	0
Ward	26	12	4	0
Recovery room	3	1	1	0
X-ray	2	1	0	0
Total	148	63	39	12

TABLE 2

Age	No. of Patients	Responded	Lived 24 Hours	Survived
yr.				
Up to 10	24	11	6	1
11 to 20	10	2	1	0
21 to 30	19	4	1	0
31 to 40	28	9	4	2
41 to 50	24	14	10	3
51 to 60	20	11	8	1
61 to 70	16	10	7	4
71 to 80	7	2	2	1
Total	148	63	39	12

TABLE 3

Group	No. of Patients	Responded	Lived 24 Hours	Survived
1. Operative	94	46	32	12
Elective	29	22	17	10
Emergency	34	16	11	2
Trauma	31	8	4	0
2. Postoperative	17	9	4	0
Elective	5	5	2	0
Emergency	7	3	2	0
Trauma	5	1	0	0
3. Nonoperative	37	8	3	0
Acutely ill	15	4	1	0
Trauma	22	4	2	0
Total	148	63	39	12

There were 12 survivors from 94 cases of arrest that occurred during an operation. Almost one-half responded; and one-third lived for at least 1 day. These included elective procedures as well as emergency operations for trauma and acute disease processes.

In the postoperative period 17 arrests occurred. Although the response to resuscitation was almost as good as that in the operative group, none survived.

Out of 37 cases in the nonoperative group, there were no survivors. These cases were primarily traumatic or terminal.

OPERATIVE GROUP

Anesthesia. Ninety-four arrests were associated with an operation. Except for minor procedures carried out on 7 in either the emergency clinic or the ward, all took place in the operating room. In 22 cases of arrest in patients so severely ill or critically injured as to require no anesthesia, only 2 responded; and the longest survival was for 8 hr. Nine of 53 patients survived arrest when the procedure was performed under general anesthesia. There was 1 arrest with spinal anesthesia, but 2 others occurred at the time of general anesthetic induction when the spinal level proved inadequate. There were no survivors from 3 arrests that occurred during hypothermia. Five of 6 hearts responded that had arrested during topical anesthesia, but there were only 2 survivors. None of 7 patients survived an arrest that occurred during a local anesthetic.

Vagal factors. Of the 94 arrests which occurred during operation, a vagal reflex could be definitely incriminated in 26 (table 4). All of these had either an ineffective beat or an asystole. In 13, a transoral procedure was being performed, whereas 10 others had their origin during manipulation of abdominal viscera. In 47 cases, the

TABLE 4

Operative Group	No. of Patients	Ineffective Contraction	Asystole	Systole	Ventricular Fibrillation	Not Stated
Vagal	26	5	19			2
Inadequate atropine	47	8	32	3	3	1
Anesthesia	32	4	22		3	3
Blood loss	37	4	17	11	3	2
Anoxia	28	4	19	1	1	3
Drug reaction	6	1	5			
Irritation	13	2	8	2	1	
Motion	9	4	5			
Electrolyte imbalance	6		5		1	
Heart disease	28	4	16	4	2	2

majority of which fell into the category of vagal reflex, the atropine given as premedication was either omitted or inadequate in amount; 40 of these were recorded as ineffective beat or asystole.

Blood loss. There were 37 cases in which blood loss was a factor. In 10, excessive bleeding had occurred before surgery; in 12, there had been unreplaced blood loss during surgery; and in 15, both occurred. None of the 11 patients with arrest in systole survived.

Anoxia. Anoxia was present in 28 cases. In 22, pulmonary function had been altered by prior chest disease, the surgical procedure, trauma, and/or severe blood loss. In 1 case the endotracheal tube had been inserted into the esophagus, whereas in another the tube was well down into the right main stem bronchus, thus occluding the left bronchial tree. Mucous plugs, blood clots, or foreign bodies were occluding the airway in the remainder.

Drug reaction. A drug reaction was present in 6 cases. Four of these were having transoral endoscopy under topical anesthesia, with Cyclaine in 3 and cocaine in 1. In the other 2 cases, critically ill patients, minor surgical procedures were being performed under local block with procaine hydrochloride. All of these arrests occurred either in diastole or with an ineffective beat.

Irritation. Direct irritation of the heart and great vessels played an undetermined role in 13 cases. The majority were associated with either trauma or a dissecting aneurysm. There were 4 cases of cardiac tamponade, with 2 in systole secondary to rupture of a dissecting aneurysm into the pericardium. The other 2 were cases of penetrating trauma, 1 occurring in systole.

Passive motion. Of great interest was the number of arrests which took place while the patient was being moved. There were 9 in this group, and all arrested in either diastole or with ineffective beat.

Other factors. There was an electrolyte imbalance in 6 patients, of whom 5 were in acidosis. Previous heart disease was present in 24. Of the latter group, 6 were cases of congenital cyanotic defects; and the remainder had either hypertension or some form of coronary artery disease.

POSTOPERATIVE GROUP

Anoxia. In the postoperative group of 17 cases, anoxia was incriminated 12 times as the major cause of arrest (table 5). The 11 described hearts arrested in diastole. Of these cases 8 were the result of respiratory arrest, which had gone unnoticed in the poorly or nonreactive patient.

Other factors. Moving the patient, either to or from the stretcher, was associated with arrest in diastole 3 times. The other factors were blood loss, vagal reflexes, electrolyte imbalance, and a penicillin anaphylaxis.

NONOPERATIVE GROUP

Anoxia. Among the 37 nonoperative cases, anoxia was a frequent predisposing factor (table 6). This was present in 14 cases. Of these, 10 were due to respiratory arrest, secondary to intracranial trauma or an overdose of sedatives. Airway obstruction occurred in 4. Altered pulmonary function, as a result of trauma, excessive blood loss, or chronic lung disease, was present in 19 other cases not included in this group. Except for the cases of trauma, all described arrests were in diastole or had an ineffective beat.

Other factors. Blood loss played a role in 20 instances, 18 being from acute hemorrhage. There were 2 arrests in systole. Other factors were an electrolyte imbalance and the vagal reflex. Cardiac disease was present in 7 patients, of whom 5 had cardiac tamponade and 1 had an acute coronary occlusion.

TABLE 5

Postoperative Group	No. of Patients	Ineffective Contraction	Asystole	Systole	Ventricular Fibrillation	Not Stated
Vagal	2		2			
Blood loss	3	2	1			
Anoxia	12		11			1
Electrolyte imbalance	2		2			
Drug reaction	1				1	
Motion	3		3			

TABLE 6

Nonoperative Group	No. of Patients	Ineffective Contraction	Asystole	Systole	Ventricular Fibrillation	Not Stated
Vagal	7	3	3			1
Blood loss	20	3	10	2	2	3
Anoxia	14	2	7			5
Electrolyte imbalance	8	1	6			1
Drug reaction	1					1
Cardiac disease	6		3	2	1	

TYPE OF ARREST

The arrest was diagnosed as an ineffective beat in 19 cases (table 7). Although the heart responded to massage in 13, there were but 6 survivors. The other 6 successful cases were in a group of 92 who had arrest in diastole. There were no survivors in any of the other categories. Among the 15 patients in whom the type of arrest was not diagnosed there were no survivors. This exemplifies how lack of familiarity with the resuscitative procedure almost precludes success.

MANAGEMENT

There were many limitations in management which may have altered the eventual course. One patient had cardiac massage for 15 minutes before instigation of controlled pulmonary ventilation. In 3, ventricular fibrillation was produced by an excessively large dose of intracardiac Adrenalin; and in 4, many different drugs were used without allowing sufficient time for any one to become effective. There was massage without blood transfusion in 29 cases, even though the resuscitator noted a poorly filling heart and absence of palpable carotid pulsations in spite of energetic manual systole.

INFECTIONS

Before closure of the chest, routine prophylaxis has been irrigation of the pleural space and thoracotomy wound with 250,000 to 1,000,000 units of aqueous penicillin followed by postoperative maintenance with both penicillin and streptomycin. This was done in all but 5 of the 23 patients who lived 3 or more days. Of the 5 who did not receive the protective antibiotic routine, 4 had infectious complications; 2 other patients developed infections despite prophylactic antibiotics. There were 4 cases of empyema and 2 of wound abscess. Cultures in 5 grew hemolytic *Staphylococcus aureus*.

Wound infection, either as empyema or as a wound abscess, can prove to be a serious complication of cardiac massage. There was 1 death secondary to empyema in a successfully resuscitated patient. Antibiotic prophylaxis is an important detail of cardiac resuscitation and should not be omitted.

TABLE 7

Cardiac Status	No. of Patients	Responded	Lived 24 Hours	Survived
Ineffective contraction	19	14	12	6
Asystole	92	44	26	6
Systole	13	3	0	0
Ventricular fibrillation	9	0	0	0
Not stated	15	2	1	0
Total	148	63	39	12

LATE DEATHS

There were 51 patients who initially responded but later died of complications from the arrest itself or from the resuscitative procedure (table 8). Primarily, a neurologic deficit was the major factor responsible for death in 33 of 35 instances in which it occurred. Repeat cardiac arrests accounted for 8 deaths, and all but 1 of these occurred within 8 hours. Although there have been reports of survivals from a second arrest, in general this has a grave prognosis. Cardiac deterioration and renal damage were among other complications. Often there was pre-existing cardiac disease; and arrest with its postresuscitative cardiac complications was merely an accelerated phase of the process. Renal damage consistent with anoxic nephrosis was present in 5 patients, 3 of whom died from this specifically. Close scrutiny should be made of fluid and electrolyte intake, as well as urinary output, in the several days that follow arrest. The lives of 2 patients might have been saved had renal malfunction been recognized earlier. Pulmonary complications led to death in 2 patients, but these were secondary to the original operative procedure. Empyema, developing directly from the resuscitating thoracotomy, was responsible for 1 death.

SURVIVORS

In 11 of the 12 survivors the heart responded promptly to massage alone within 1 minute. Ten minutes of massage and a single intracavitary injection of Adrenalin were necessary to produce an effective beat in the 12th. Not one had a neurologic deficit, even transiently. One patient had had a cerebral vascular accident several months before the time of cardiac arrest, but he had no detectable neurologic changes. Eight had pulmonary complications; 3 developed post-

TABLE 8

Late Deaths	No. of Patients	Direct Cause of Death
Central nervous system	35	33
Pulmonary	8	2
Renal	6	3
Infection	4	1
Cardiac	11	3
Cardiac arrest	7	7
Original disease	1	1
Not stated	1	1

thoracotomy infections, 2 in the wound and 1 an empyema. Progression or onset of cardiac disease occurred in 6. No survivors had significant renal complications. At a later date 7 tolerated anesthesia without difficulty, 6 of these procedures being general and 1 spinal. Only 1 patient required a tracheotomy in the immediate postresuscitative period, and this was for management of his chronic lung disease. Six of the survivors have had progression of their original disease, and 5 of these have died.

DISCUSSION

This series is unique in that more than one-third were traumatic cases; almost one-third occurred during emergency operations, and only one-fourth were in elective procedures. This is representative of the large number of trauma cases seen at Grady Memorial Hospital and the many emergency operations performed for both traumatic and acute disease processes.

OPERATIVE AND POSTOPERATIVE CASES

There are several critical periods in the operating room when arrests are prone to occur. At these times the operating room personnel should be especially alert:

1. During induction of general anesthesia. There were 12 of the patients who fell into this category; all had prompt massage. The majority were either vagal or anoxic in nature.

2. Topical anesthesia. Reactions to topical anesthesia can be either vagal or anaphylactic. Out of the 6 in this series, 4 were vagal and 2 anaphylactic. Unfortunately, the sensitivity reactions were not recognized and massage was done without any attempt to reverse the peripheral vascular collapse Of the 6 patients, 5 were treated with barbiturates at the onset of arrest, under the assumption that these were neurologic reactions to the cocaine or cocaine-like drugs. As a result, cardiac massage was delayed in 2 cases. None of these patients received appropriate atropine or Adrenalin.

3. Change in method of anesthesia. Cardiac arrest occurred at the time of induction of general anesthesia in 2 patients who initially had spinal anesthesia, but in whom length of operation or inadequacy of spinal level forced the use of a general agent. There was 1 survival. Atropine had not been administered preoperatively to either of these patients, and the predisposing factors seemed to be vagal in nature.

4. Vagal reflexes. There were 33 instances of arrest during periods when vagal afferents received considerable stimulation. These were during tracheal intubation, during esophageal, tracheobronchial, or oral procedures, and during visceral manipulation, especially cardiac, great vessel, or biliary. Often a premonitory period of bradycardia occurred.

There were 47 cases which fell into 1 of 3 categories: (a) insufficient dose of belladona as premedication; (b) delay in beginning anesthesia and/or operation, the premedication having been given several hours before; and (c) prolonged operative procedures without readministration of belladona.

Of these patients, 40 had asystole or ineffective beat. Probably a number of these arrests might have been prevented with more adequate suppression of the vagal reflex.

Bradycardia was a premonitory sign in 27 cases (table 9). In 18 of these atropine premedication was inadequate in amount or timing. Two of these patients were given intravenous atropine at the onset of bradycardia. In both,

TABLE 9

Category	No. of Patients	Ineffective Contraction	Asystole	Systole	Ventricular Fibrillation	Not Stated
Bradycardia	27	5	21	0	0	1
Anoxia	54	6	37	1	1	9
Hypovolemia	60	9	28	13	5	5
Motion	12	4	8	0	0	0

atropine corrected the bradycardia; but each arrested 2 and 3 hours later after returning to the ward. Not included in this series are 2 patients who developed bradycardia. Each was given atropine intravenously and neither arrested. One was during exploratory thoracotomy for a laceration of the left subclavian artery, and the other was a case of hypovolemic shock from multiple pelvic fractures.

The above brings out 4 important points: (a) atropine should be given preoperatively in adequate amounts and repeated as necessary; (b) bradycardia may be a premonitory sign of cardiac arrest; (c) atropine should be given intravenously when bradycardia develops and readministered as necessary; and (d) anoxia can cause bradycardia, and adequate pulmonary ventilation with oxygen therefore must be assured.

5. Anoxia (*table 9*). There were 54 patients with moderate to severe anoxia at the time of arrest; 2 of these had a definite bradycardia before arrest. Airway obstruction was responsible in almost all instances.

Anoxia, however, did not prevent successful resuscitation, as the 6 survivors proved. As long as immediate pulmonary ventilation with oxygen accompanies active cardiac massage, there should be a fair chance for success.

6. Excessive hemorrhage. There were 25 cases in which arrest occurred at the time of massive blood loss without prompt replacement or control of bleeding. This certainly indicates that unless blood can be replaced rapidly or blood loss controlled, massage is almost useless. None of the patients who fell into this category survived.

Sixty cases of arrest occurred in association with significant hypovolemia (table 9). None of these patients survived massage. The hearts responded in only one-fourth, and 7 lived little longer than a single day. All 13 systolic arrests from the entire series were in this group.

Despite the Trendelenburg position and obstruction of the thoracic aorta, significant hypovolemia could not be overcome. Blood replacement was never adequate or rapid enough to give an effective circulating volume; and massage could produce no more than a weak carotid pulse. Although manual systole is being carried out and although pulmonary ventilation is oxygenating the blood, a relatively normal blood volume is required for ventricular filling and cerebral profusion. All patients who responded initially were decerebrate from the beginning. Thus, the presence of an effective circulating blood volume, or the expectancy to have such within 5 minutes of arrest, is a prerequisite for successful resuscitation. This not only involves rapid and massive transfusion, but also immediate control of major bleeding sites. At present, rarely if ever can such be accomplished within so brief a period of time.

7. Motion. Passive motion played a predisposing role in 12 cases (table 9). Nine were instances in which the patient was moved to or from a stretcher during the immediate postoperative period. The other 3 occurred at the time of applying a large dressing. Several of these arrests could certainly have been averted with more gentle handling of the lethargic or anesthetized patient.

8. Prolonged anesthetic effect. There were 9 arrests which occurred on the ward in patients who undoubtedly had respiratory arrest secondary to a persistent effect of the anesthetic agent. None of these was salvaged. The premature removal of a nonreactive patient from the operating area is poor judgment unless the facilities of a recovery room are available.

TRAUMA

The extent of trauma can be roughly correlated with degree of hypovolemia in most instances. There were 58 patients with trauma, with 13 to respond, 6 to live for 24 hours, and no survivors. All who responded became decerebrate. If all cases of recent trauma or hemorrhage are considered, a similar correlation to hypovolemia can be made. There were 109 patients who had had recent operative or nonoperative trauma or in whom there had been recent hemorrhage. Only 3 survived, and these 3 exhibited no abnormalities that would indicate a reduced blood volume.

Probably a more accurate evaluation of the relationship between trauma and hypovolemia can be drawn from contrasting the level of shock with the response to massage. In 52 patients who had sustained trauma, some degree of shock was noted. In general, a more profound depth of shock prior to arrest resulted in a lower incidence of initial response and a shorter period of survival.

In summary, it can be categorically stated that cardiac massage offers little hope of success in the face of significant hypovolemia, severe

trauma, or massive uncontrolled bleeding. Thus, a third requirement for successful cardiac resuscitation can be added to the well publicized two: (1) effective manual systole, (2) pulmonary ventilation with oxygen, and (3) production and maintenance of an effective blood volume.

TYPE OF ARREST

Not only is it necessary to satisfy these 3 requirements, but also the specific factor or factors which led to arrest must be rectified. In this way, optimal conditions are established for the heart to respond. Vagal reflexes should be suppressed, major bleeders controlled, the blood volume expanded, or anaphylaxis treated. Otherwise, energetic attempts might prove futile.

The status of the heart at the time of thoracotomy can give considerable insight into the predisposing factors. Arrest in systole indicates moderate to severe hypovolemia and an essentially hopeless situation. Although none of the 9 fibrillating hearts could be defibrillated, this is not hopeless, as can be verified from the literature. When the resuscitating team could not recognize the status or importance of a particular type of arrest, their lack of familiarity with the procedure was reflected in a lack of success. The ineffectively beating heart and its end stage of diastole represent the best chances for salvage. These are usually cases of vagal reflex, anoxia, cardiovascular irritation, or anaphylaxis.

SUMMARY

This is a study of 148 patients who underwent cardiac massage at Grady Memorial Hospital during the 7-year period beginning July 1, 1952. Analyses of response and survival were made with respect to age, area in the hospital where arrest occurred, condition of the patient before arrest, various predisposing factors, and the status of the heart at the time of thoracotomy. There were no survivors from cardiac arrest which occurred outside of the operating room. No patients who were seriously ill, or who had sustained recent nonoperative trauma, were resuscitated with permanent success. Vagal reflexes, insufficient atropine premedication or failure to readminister the drug, induction of anesthesia or change in the method of anesthesia, postoperative oversedation, hypovolemia, anoxia, allergic reactions, and external physical stimulation of the lethargic patient were the major predisposing factors. An ineffective beat with prompt response to massage offered the greatest chance for survival. No patient survived a second arrest; nor were there any survivors who had been in shock, no matter how mild, before the occurrence of arrest. The futility of massage in the face of hypovolemia and the impending cardiac arrest indicated by bradycardia are stressed. The use of atropine to prevent or treat this bradycardia is encouraged. With respect to the survivors, cardiac deterioration, post-thoracotomy infection, and risk of subsequent anesthesia are mentioned.

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RECONSTRUCTION OF NORMAL URINARY CONTINUITY AFTER URETEROILEAL BLADDER DIVERSION*

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Segments of terminal ileum have been used to replace the ureter for maintaining continuity to an intact bladder,[5, 7, 12] for enlargement of a contracted bladder,[14, 17] and for total external urinary diversion.[2] Its application in various phases of genitourinary and gynecologic surgery has been detailed in a previous communication.[10] There are many cases in which plastic repair of congenital anomalies or acquired defects of the bladder and urethra might be successful if the urinary flow were totally diverted. It would be desirable, of course, to reconstitute the urinary flow system after the repair is completed.

Temporary diversion of the urinary flow to an isolated urinary ileal bladder (conduit) with subsequent reconstruction of the integrity of the normal urinary flow system is the subject of this experimental study. The physiologic response to the stepwise procedures over a long follow-up period is reported.

METHOD

In 10 healthy female mongrel dogs weighing 20 to 35 lbs., an ileal bladder was created from a 6- to 12-cm. segment of terminal ileum after the method of Bricker.[4] The distal ureters were ligated near the bladder. After a period of 3 to 8 weeks a second operation was performed in which the urinary ileostomy was closed and the distal end of the urine-carrying ileal segment anastomosed to the dome of the resting, contracted bladder. If any narrowing of the ureteroileal anastomosis was present at this operation it was mechanically dilated. The ileal segment and the bladder were flushed and filled with 0.5 per cent neomycin sulfate solution at this operation, and penicillin, streptomycin and 0.5 per cent neomycin were placed in the free peritoneal cavity.

Follow-up studies of the status of the urinary tract integrity were made with periodic excretory pyelograms in all animals. Retrograde cystograms were also made. Renal function was followed with serial blood urea nitrogen and creatinine determinations. The physiologic response was determined by following the general condition and weight of the dog, together with serum carbon dioxide content, chloride, sodium, and potassium. Determinations of these values were also made in a control series of animals. All dogs were maintained on a high protein kennel diet. Routine postoperative care was given.

RESULTS

Table 1 gives the results of excretory pyelographic and retrograde cystographic studies on these preparations. Abnormal findings were quite common immediately after ileal bladder diversion due to stricture of the ureteroileal anastomosis. After dilation at the second operation this was remedied, and subsequently most pyelograms were normal. One dog (no. 3) developed a marked hydronephrosis on the left from what appeared by pyelographic study to be a stone at the ureteroileal junction. Two animals died early before studies were undertaken.

Typical of the excretory pyelographic and retrograde cystographic studies are figures 1 and 2. Note the filling of the ileum in the excretory pyelograms. In figure 2*B* it can be seen that only the left ureter filled with the cystographic study. Similar unilateral retrograde filling occurred in 2 other animals. Two dogs had no reflux into either ureter, and in 3 dogs only the bladder was filled with dye. Immediate excretory pyelograms in all dogs showed that in all cases except 1 (dog 3) this was due to a valve action at the ureteroileostomy and not to stenosis.

Results of gross renal function evaluation by serum creatinine and blood urea nitrogen determinations are shown in figure 3. Note that the

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TABLE 1

Excretory pyelograms

Dog No.	Pyelogram after Ileal Bladder		Pyelogram after Ureteroileocystostomy				Cystographic Reflux	Time Followed
			<4 mo.		>10 mo.			
								mo.
1	+	++	N	+	N	+	Bladder only	13
2	N	N	*		*		*	4
3	N	N	N	++	N	++	Bladder, ileum	15
4	++	++	+	+	N	N	Bladder only	15
5	++	++	N	N	N	N	Bladder, ileum, left ureter	10
6	++	+	N	N	N	N	Bladder only	14
7	+	+	*		*		*	1
8	N	N	N	N	N	N	Bladder, ileum, right ureter	13
9	N	N	N	N	N	N	Bladder, ileum, left ureter	10
10	+	N	N	N	N	N	Bladder, ileum	12

Key: N = normal; + = moderate hydronephrosis; ++ = marked hydronephrosis; * = dead.

FIG. 1. Dog 5. *A* (*left*), excretory pyelogram 3 months after ileocystostomy. Note the dye in the ileum. *B* (*right*), retrograde cystogram together with excretory pyelogram. Before the intravenous dye only the left ureter filled.

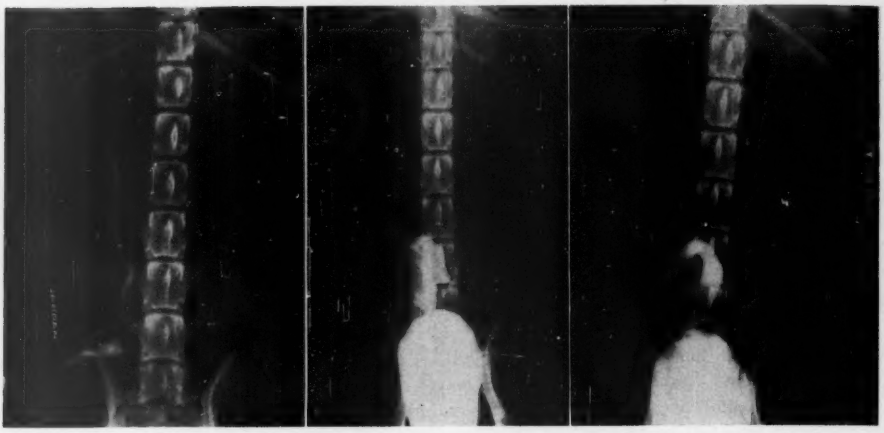

FIG. 2. Dog 9. *A* (*left*), excretory pyelogram 9 months after ileocystostomy. *B* (*middle*), retrograde cystogram. Note that only the left ureter has refluxed dye. *C* (*right*), the same study after an excretory pyelogram. Note that the right ureter and pelvis are normal.

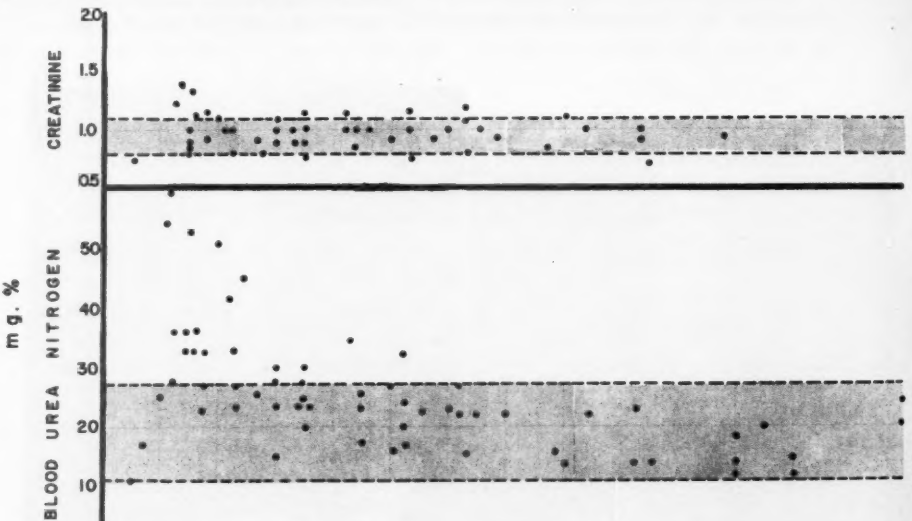

FIG. 3. Studies on renal function. Values are plotted for all dogs. The normal range is outlined with *stippling*. Time is after first procedure.

blood urea nitrogen values were only high immediately after the ileal bladder diversion. With correction of stenosis at the ureteroileal junction the values returned to, and remained, normal. Similar results were seen with serum creatinine determinations.

No gross deviation from the normal control range was seen with serum carbon dioxide content, chloride, sodium, and potassium (figure 4).

All animals prospered and gained weight. Two dogs died before being studied, as noted above. The cause of death was obstruction of the stoma

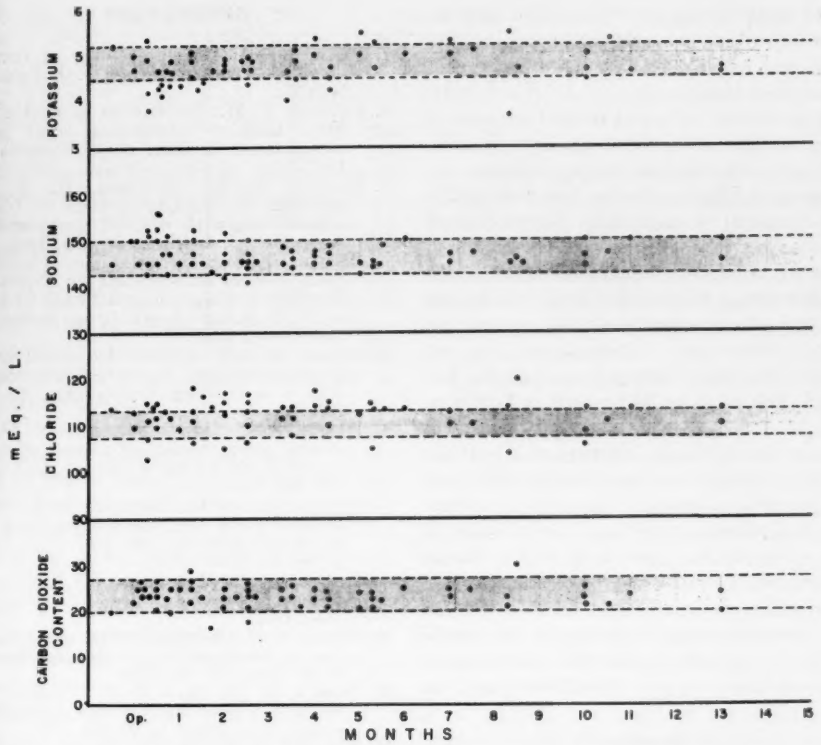

FIG. 4. Studies on the physiologic response. Values are plotted for all dogs. The normal range is outlined with *stippling*. Time is after the first procedure.

between the ileum and bladder in 1 case, and anesthesia at the time of ileocystic anastomosis in the other. Three dogs died at 8, 9 and 11 months. One of the latter deaths was associated with delivery of a litter of pups. The other 2 were due to wounds from dog fights. The remaining 5 animals were sacrificed after 13 to 15 months.

DISCUSSION

Follow-up of up to 15 months on the experimental animals reported here has revealed no uncontrollable problems. That the procedure can be safely performed is evident. The use of segments of ileum for ureters has been reported, both experimentally,[6, 11, 19] and clinically.[1, 8] The use of a segment of terminal ileum as a conduit for urine has received wide acceptance.[3, 13] It is only logical that the 2 procedures might be beneficially combined.

We previously reported the planned use of temporary diversion to an isolated ileal bladder in the correction of an intractable vesicoureterovaginal fistula.[10] This 47-year-old white woman, 2 years after operative reconstruction of urinary tract continuity, has continued to do well.

Shoemaker[15] in 1906 first performed a temporary diversion of urine to an ileal bladder. The patient was an 18-year-old female with probable tuberculous contracture of the bladder. Diversion to an isolated ileal segment was performed with anastomosis of the one remaining good ureter. After 1½ years, because of severe skin irritation about the urinary ileostomy, the ileal conduit was attached to the contracted bladder. He reported the patient to be alive and symptomless 1½ years later.

Unplanned temporary diversion was also used by Ulm.[18] He reconstructed the urinary tract of a 24-year-old man who had had ileal bladder diversion for a hypotonic, neurogenic bladder. His procedure was to implant the ileal loop in the

bladder after cystoscopic examination had revealed it to be in apparently good condition. The patient was reported to have done well except for rare stress incontinence.

The possibility for use of an ileal segment to salvage bladder continence should be considered. Innervation of the bladder is a prerequisite.

Exstrophy of the bladder has long been a difficult problem in reconstruction. The method of treatment has varied. Strong opinions have been voiced for permanent urinary diversion to the sigmoid or ileum with excision of the bladder and correction of any epispadias,[9] and also for attempted plastic repair.[16] Primary repair has not been recommended, largely because of the low survival rate of 40 to 50 per cent in long term follow-up and the frequent renal morbidity in the survivors.[9] Plastic reconstruction of the bladder would be much more feasible with total safe, reversible diversion of the urinary stream. In such a situation a prolonged period could be allowed for healing, growth and even plastic enlargement of the bladder, if necessary.

Temporary diversion might be used also for more adequate surgical treatment of vesicovaginal fistulas and for any other condition for which the bladder needs to be put temporarily at rest.

SUMMARY

Long term follow-up studies were performed on 10 dogs in which temporary diversion of the urinary flow had been made to an isolated ileal loop with subsequent reconstruction of the urinary tract continuity by a ureteroileocystostomy. Retrograde cystographic and excretory pyelographic studies showed good retention of the integrity of the upper and lower urinary systems. Serum creatinine and blood urea nitrogen studies were normal after some early deviations. Blood electrolytes remained largely within control limits throughout. It was concluded that temporary urinary diversion to an isolated ileal loop with subsequent reconstruction of the urinary tract by an ileocystostomy might have great value in plastic repair of congenital anomalies or acquired defects of the lower urinary tract.

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NASOGASTRIC SUCTION: A REVIEW

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The nasogastric tube and its role in the postoperative care of the surgical patient has come under close scrutiny in the past few years. Although historically, the nasogastric tube evolved as a means of introducing substances into the stomach, in modern surgery it is used more extensively to empty the stomach of gases or fluids. The value of nasogastric suction in the routine care of the postoperative patient has recently received critical appraisal.

Certain indications for the use of the nasogastric tube and nasogastric suction are not disputed. It aids in diagnosis, *e.g.*, gastric secretory analysis in peptic ulcer, and serves as a conduit for the exfoliated cells which are examined in the detection of gastric malignancy. When pyloric obstruction is present with subsequent gastric retention, the stomach may be emptied through the tube. Patients unable to take oral feedings may at times be nourished by tube feedings. It is recommended as a therapeutic adjunct in the treatment of acute pancreatitis; removal of gastric acid will decrease pancreatic secretion by curtailing secretin production. Also, whenever *actual* gastric distention is present, preoperatively, during surgery or postoperatively, the use of nasogastric suction will effectively ameliorate this condition.

By far the most common use of nasogastric suction is a prophylactic one, in an attempt to prevent postoperative intestinal distention. This use has resulted from those experiments demonstrating that swallowed air is the offending agent in postoperative distention. Wangensteen[10] proved that the use of nasogastric suction to remove swallowed air would largely eliminate this danger. Subsequently, most modern surgeons have been trained to use nasogastric suction prophylactically.

Postoperative intestinal distention manifests itself as an adynamic ileus and rarely as acute gastric dilation. Ilgenfritz[6] states that "acute gastric dilatation is often noted in conjunction with the paralytic intestinal obstruction characteristic of generalized peritonitis, although it is relatively frequent also following operations upon upper abdominal viscera." The entity results from the rapid accumulation of large quantities of fluid and gas in the stomach.

During an adynamic ileus, swallowed air accumulates and distends the intestines. In the process of deglutition, relaxation of the superior esophageal constrictor permits the bolus of food or air to pass from the pharynx into the stomach. The negative intraesophageal and intragastric pressures produced by breathing with the glottis closed can force 50 to 160 cc. of air through a relaxed superior esophageal constrictor.[7] In such a manner 1 to 2 L. of air may accumulate in 3 to 5 minutes. In most people the constrictor is closed except during swallowing. However, a nasogastric tube prevents tight closure of this sphincter. It can be seen, therefore, that the tube may increase the amount of air swallowed and hence increase the distention. Mehnert and co-workers[8] have shown that nasogastric tubes do not remove all intragastric secretion or gas.

The role of air swallowed during the induction of anesthesia reportedly has little effect on postoperative distention. Maddock and associates[7] determined that an average of about 15 cc. of air was swallowed by 23 patients during induction. A cuffed endotracheal tube aids in preventing excess air or gas from entering the stomach.

Of secondary importance in the cause of postoperative intestinal distention is retained secretions. With peristalsis absent one might think that the intestinal secretions would pool and increase distention. However, secretion as well as peristalsis is diminished postoperatively.[2]

Recently, several groups of investigators have chosen to evaluate the postoperative treatment of surgical patients both with and without nasogastric suction. Eade and others[2] used no suction at all in 130 out of 143 consecutive major abdominal procedures, including subtotal gas-

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trectomy, cholecystectomy, choledochostomy, colectomy and hysterectomy. The patients were cautioned not to swallow anything and were given nothing by mouth until flatus was passed. Morbidity was slight and there was no mortality. Gerber and co-workers[4] described 1000 consecutive cases of paralytic ileus treated without nasogastric suction. These included all types of stomach, biliary tract and colon surgery, traumatic abdominal wounds and peritonitis. Oral intake was prohibited to these patients. The return of peristalsis was not delayed by this method of treatment. Acute gastric dilation was not seen in this group of 1000 patients.

Mehnert and associates[8] also attempted to evaluate the convalescence of patients who were intubated and permitted to take water while the nasogastric tube was in place. In a control group of 10 postoperative patients who were fasted while intubated, about 600 cc. of gastric fluid and 2000 cc. of air were removed from each patient in 24 hours. In a comparable group of 17 patients who were allowed to drink up to 3 L. of water each 24 hours, there was no appreciable increase in intestinal secretions. Mehnert observed that these intubated patients trapped 1.3 cc. of air with each 1 cc. of water they swallowed. The patients who drank 3 L. of fluid did have more air in their stomachs but still had no difficulties with distention. Mehnert concluded that the average postoperative patient who normally swallows 2 L. of air and 600 cc. of recoverable secretions can manage the additional air accumulated by drinking moderate amounts of fluids.

Nasogastric suction is not wholly innocuous. Injuries to the nose, laryngeal stenosis and stricture, vocal cord paralysis, esophageal stricture, and stomach and esophageal perforation have been associated with stomach intubation.[1, 2] Difficulties arising from electrolyte imbalance subsequent to nasogastric suction are not uncommon.[9] In regard to the control of postoperative nausea and vomiting, Mehnert[8] stated that 45 per cent of his patients had nausea and 20 per cent vomited while the tubes were in place and functioning well. Gerber and co-workers[4] examined two series, each composed of 300 consecutive cases of paralytic ileus; one series of patients was intubated, the other was not intubated. In the intubated control group, 17 of 56 patients (30.3 per cent) who underwent biliary tract surgery vomited. In the nonintubated group, 21 of 71 patients (29.5 per cent) vomited. In spite of intubation there was no difference in the incidence of vomiting. All surgeons are familiar with the common complaints of sore throat, tube discomfort and thirst voiced by patients with nasogastric tubes.

In an attempt to bypass the hazards of the nasogastric tube but still use suction as prophylaxis against postoperative intestinal distention, Farris and Smith[3] and Gilchrist[5] advocate temporary gastrostomy performed at the time of surgery. Using either a Levin tube with additional perforations or a Foley catheter with 10 cc. of water to inflate the bulb, these authors report safe, comfortable control of intestinal gas and fluids. The tube or catheter is withdrawn when peristalsis resumes; subsequent intraperitoneal leakage was not seen in the 72 patients reported by Farris and Smith. However, both authors recommend temporary gastrostomy only in aged or debilitated patients or those with special complications such as poor cardiac reserve, bronchiectasis, and others. Routine prophylactic intragastric suction in all postoperative patients, whether by nasogastric tube or temporary gastrostomy, is not advocated.

SUMMARY

In large series of patients who underwent varied types of abdominal surgery, postoperative intestinal distention necessitating nasogastric suction was quite rare. It is clear that most postoperative patients can be managed successfully without this procedure. One cannot deny, however, that some patients will develop intestinal distention after surgery and will need suction. These patients cannot be identified ahead of time, therefore careful postoperative observation and evaluation and use of nasogastric suction only when distention is present should be the practice. Actual distention and not its possible occurrence is the valid indication for postoperative nasogastric suction.

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PERFORATING CARCINOMAS OF THE COLON

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With the advent and widespread use of antibiotics for the treatment of infections of all types, the surgical management of perforating lesions of the colon has been greatly enhanced, especially the acute free perforations with generalized peritonitis and the slower localized perforation of the growing neoplasm.

There is a paucity of articles on the subject of perforating carcinomas. Before the advent of the sulfa preparations in the late 30's and the antibiotics in the early 40's, approximately 100 per cent of these patients with free perforations died, whether they were subjected to laparotomy or not. The largest group reviewed was by Donaldson[3] in 1958. He studied 182 cases of perforating carcinomas of the colon at the Massachusetts General Hospital, and found this to represent 7.8 per cent of all the patients with carcinoma of the colon who entered the Massachusetts General Hospital over a 20-year period. In the cases reviewed, there were 39 patients with free perforations (21 per cent), 93 with localized abscess (15 per cent), and 50 with fistula formation (28 per cent). The method of treatment for these free perforations was simple drainage, bypassing or exteriorization. Two of these patients had primary excision and both succumbed. Fifty-three per cent of all patients with free perforations died regardless of the procedure undertaken.

Gregg,[4] from Syracuse, found in his own private practice that of 116 patients with carcinoma of the large bowel, 20 were operated on for acute obstruction, 4 of these having acute free perforation. No mention was made in this series of localized perforation and/or fistula.

Albers and co-workers,[1] in a study of 72 cases of cecal perforations at the Los Angeles County Hospital, found that 16 were due to carcinoma. The mortality rate in this series was 69 per cent. The surgeons of this group utilized several methods, but felt that the simplest surgical procedure was the most effective treatment in these patients; that incision and drainage of the abscess or a tube cecostomy gave equally good results. However, 6 of their patients developed fecal fistula. Eight of the 16 patients in this group were not operated upon and the mortality rate was 100 per cent. There were 2 surgical deaths.

This discussion will deal with 80 unselected cases reviewed in the East Tennessee Baptist Hospital during the period from 1949 to 1959, and in the University of Tennessee Memorial Hospital and Research Center, which has been functioning only for the past 3 years. The histories, operative reports and pathologic studies have, of necessity, been the basis of evaluating the type of disease present, the extent of the disease, and the general condition of the patient and of the peritoneal cavity. It is well to state that these patients were treated by 18 different surgeons, the majority of whom are well trained.

The distribution of the 80 cases of carcinoma reviewed is shown in table 1.

For this study of the perforating or perforated carcinomas of the colon, they were divided into three groups: (1) free perforation of the colon with generalized peritonitis; (2) perforation with localized abscess; and (3) perforation with fistula. It is well to state that the majority of these patients with free perforation had complete bowel obstruction. Others were found to have the perforation with localized abscesses and only low grade obstruction at operation. Also, other patients were found with perforation and fistula at the time of the original operative procedure. In accord with the majority of the authors of the papers on the subject reviewed, it is felt that the type of procedure followed in the treatment of a perforating or perforated carcinoma will depend entirely upon the extent of the perforation, the extent of the disease, the condition of the bowel and the patient in general (table 2).[5, 6]

FREE PERFORATION

The operative procedure of choice for the 11 cases of perforations with generalized peritonitis was that of exploration for an acute abdomen, a venting procedure being performed. Three of the

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TABLE 1

Distribution of 80 cases of carcinoma

Site	No. of Cases	No. Perforated
Sigmoid	46	23
Descending colon	7	2
Splenic flexure	4	4
Transverse colon	5	4
Hepatic flexure	5	2
Right colon	7	2
Cecum	6	4

patients had sigmoid colostomies with drainage of the pelvis, 1 had a transverse Mikulicz colostomy, 1 patient had a cecostomy, and 2 had only appendectomy and were subsequently operated upon by different surgeons. At the time of the original operation, 1 of the patients on whom an appendectomy was done was explored, and a splenic area perforation was not discovered. This patient was subsequently operated on, at which time he was found to have a localized abscess about the lesion. Another of the patients was operated on 10 days after an appendectomy, at which time he was found to have a localized abscess formation in the descending colon; a colostomy was performed, with a subsequent definitive resection. One patient had a free perforation of the splenic flexure, and only a colostomy was done because of the extent of the disease. There was 1 hospital death in this group of 11 patients. However, the late follow-up on these cases has been extremely discouraging, with only 3 patients alive, the longest period of survival being 26 months. The majority of these unfortunate individuals had widespread disease at the time of the operative procedure, but in all cases except 1 a definitive operation was performed. It is well to state that all of these patients had tumor growing in the colostomy and/or laparotomy sites at the time of demise. The sites of free perforations were: sigmoid, 8; left colon, 1; and splenic flexure, 2. There was 1 hospital death among the patients with free perforations.

PERFORATION AND LOCALIZED ABSCESS

The sites of perforation and localized abscesses were: sigmoid colon, 11; left colon, 1; splenic flexure, 2; transverse colon, 2; hepatic flexure, 1; right colon, 2; and cecum, 4. It was felt necessary to do preliminary colostomy for obstruction in 9 patients of this group. A small percentage of them had a venting procedure after the resection if they had not had a preliminary colostomy. In the majority of patients at the time of resection, the colostomy was closed. Of the 4 patients with localized abscesses of the cecum, a primary one-stage operative procedure was done, with an end-to-end ileotransverse colostomy. One 78-year-old man had a perforating carcinoma of the right colon with fistula and abscess formation and high grade obstruction. His treatment was bypass ileotransverse colostomy. He was discharged from the hospital but died at home 2 months later. None of these patients died while in the hospital.

PERFORATION AND FISTULA

Of the patients with perforation and fistula who were operated upon, there were 8 as follows: sigmoid, 4; transverse colon, 2; hepatic flexure, 1; and right colon, 1. Of the 4 with sigmoid fistula, 2 were considered inoperable at the time of the procedure because of the extent of the disease. Transverse colostomy only was done in these 2, and they died during the course of hospitalization. Two patients with transverse colon lesions, with extension and perforation out into the wall of the stomach, had transverse colon resection and subtotal gastric resection en bloc. In 1 of these patients the gall bladder was also removed. The latter patients did quite well and are still alive, the longest period of survival being 32 months. A patient with hepatic flexure disease with multiple fistula into the stomach, duodenum and jejunum had a wide resection, but had many complications following this procedure and died

TABLE 2

Operative procedures

Site	Primary Resection	Colostomy Preliminary	Perforation Free	Local Abscess	Fistula	Colostomy Only	Deaths
Sigmoid	24	13	8	11	4	6	4
Left colon	1	3	1	1	0	0	1
Splenic flexure	3	1	2	2	1	1	0
Transverse	3	1	0	2	2	0	0
Hepatic flexure	2	2	0	1	1	1	0
Right colon	4	1	0	1	1	0	1
Cecum	7	0	0	4	0	0	0
Total	44	21	11	22	9	8	6

while in the hospital. A 17-year-old girl with widespread disease had a biopsy of a sigmoid lesion with no definitive procedure being done. She died during her hospitalization and no autopsy was granted.

All of the cecal carcinomas had primary resections, and of these 6 patients with cecal lesions, 4 developed localized abscesses. One of the patients had been operated on for appendicitis 4 months prior to the definitive resection.

DISCUSSION

From the review of these 80 cases and from my own personal experience, it is felt that the patients with free perforations and generalized peritonitis should be handled by exploratory laparotomy, preliminary colostomy or exteriorization procedure and drainage of abscess areas.

In patients with localized abscess without complete obstruction, definitive resection at the time of exploration should be performed. If the lesion is in the cecum or ascending colon, a right colectomy can be done at that time in the face of obstruction. It is felt that ileostomy or cecostomy for cecal carcinomas is not indicated. If generalized peritonitis and massive soiling are present, an exteriorization procedure should be considered.

In patients with perforating carcinomas involving adjacent organs, it is recommended that a primary en bloc resection and re-establishment of continuity of the bowel be done. However, in some of the cases reviewed this was impossible. In 2 of the cases involving the stomach and transverse colon, this produced good results. However, in the patient with involvement of the hepatic flexure, duodenum and the stomach, this terminated in disaster. Two patients with sigmoid-vesical fistulas were satisfactorily handled. These patients did have preliminary venting procedures.

It has been the general policy to prepare all patients with suspected carcinoma with antibiotics and the intestinal sulfa preparations along with purgation and mechanical cleansing. At the time of the operative procedure, if the patient has not been prepared and it is necessary to do a primary resection, as in some of the cases of cecal perforation, neomycin is placed in the bowel lumen. It is well to state that in none of the cases reviewed was neomycin placed in the general peritoneal cavity. However, it has been my personal practice to place streptomycin and penicillin solution in the abdominal cavity, and in some of the cases a polyethylene catheter was placed in the abdominal cavities for subsequent irrigation. Cultures and sensitivity studies were done in the majority of cases. I believe that in carcinoma of the colon and all abdominal carcinomas, the incision should be closed with nonabsorbable sutures and that wire retention materials should be used. I personally have had no dehiscence.

SUMMARY

Eighty cases of carcinomas of the colon have been reviewed; 44 of these patients had primary resection; 11, free perforation; 22, local abscesses; and 7, local abscess and/or fistula. Of the 80 patients operated upon, 9 had colostomy only. Three patients with fistula and far advanced disease died while in the hospital; 1 patient with a right colon lesion with fistula into the stomach and surrounding organs died; 1 patient with a left colon carcinoma with reverse intussusception died, and also 1 patient, a 17-year-old girl, who had a biopsy only. The hospital death rate was 14 per cent (6 patients).

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EXPLORATORY LAPAROTOMIES TO MINIMIZE "GEOGRAPHICAL MISSES" IN RADIOTHERAPY*

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With the general availability of supervoltage x-ray equipment and of cobalt^{60} and cesium^{137} teletherapy machines, the art of radiation therapy has reached a point at which large doses of radiation can be given with ease to any tumor of moderate size, located in any region of the body. The limiting factor in carrying out a successful treatment is no longer the tolerance of the skin to radiation, but rather the differential radiosensitivity between the tumor and the organs and tissues surrounding it. Some areas of the body are severely damaged by an amount of radiation only slightly higher than that required to treat a carcinoma; it is necessary, therefore, in planning a radiation treatment, to know accurately how far the tumor and its regional metastases extend and infiltrate. Thus, normal structures may be spared if they are not involved by the malignant process.

The inability, however, to precisely determine the extent and geographical location of the tumor is one of the more frustrating limitations in clinical radiotherapy. If precise determination is difficult when the tumor is open to visual inspection as in carcinoma of the tongue, it is still more difficult when the tumor is located in an anatomic region where the eye cannot see and the fingers cannot palpate. Here we can use ancillary exploratory means, which are in the vast majority of cases simply radiography and endoscopy. In carcinoma arising from hollow organs these methods give a fairly good indication of the extent of the tumor protruding within the lumen. The part of the tumor infiltrating into the neighboring structures is, however, hardly ever delineated. The situation is still more troublesome when the tumor arises from organs and tissues that do not lend themselves so readily to x-ray or distant vision examination. Here the most important piece of information at times may be a knowledge of the natural history of that particular variety of tumor.

This state of affairs means in a practical sense that when the radiotherapist starts thinking about how to treat a patient, he makes first of all a silent wish—to guess correctly the extent of the tumor.

The following example may serve to emphasize this point: Eight months before being sent to us for radiation therapy, a 58-year-old woman was diagnosed as having a grade 3 transitional cell carcinoma of the urinary bladder. In the interval she had been treated with fulguration on two different occasions. A partially necrotic tumor, occupying the left and posterior walls of the bladder, was visible on cystoscopy. On pelvic examination a mass could be felt slightly to the left of the midline. Intravenous pyelograms showed a normal right kidney; the left one was not functioning. All the necessary pretreatment studies had been done, and yet we lacked very important information: how large the tumor really was, how far it extended to the left, what lymph nodes were involved.

Instead of relying almost entirely on indirect information, it seems logical that whenever possible the tumor should be surgically exposed by means of a laparotomy, so that its limits can be more accurately determined by inspection and palpation. This eliminates not all, but a good part, of the guessing game. Microscopic peripheral projections of the tumor will still remain unnoticed, but there is nothing one can do about these at the present time.

The patient described above was subjected to an exploratory laparotomy (figs. 1 and 2). The tumor was seen at the serosal surface at the posterior bladder wall, just crossing the midline. The infiltration did not extend much beyond the anatomic limits of the bladder on the left side. The sigmoid colon was attached to the bladder by neoplastic adhesions. The left ureter was dilated. No enlarged nodes were found along the common iliac or above the aortic bifurcation.

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* From the Medical Division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tennessee, under contract with the United States Atomic Energy Commission.

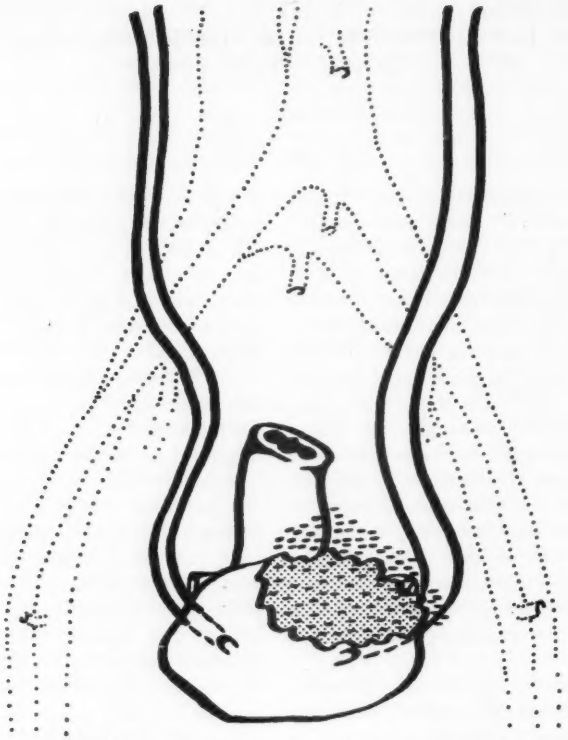

FIG. 1. Diagrams showing position and extent of a carcinoma of the bladder.

The tumor, although large, could be encompassed in a field of radiation measuring 7 by 9 cm.; the radiation beam was swung around the tumor through a 180° arc of rotation.

Although careful observation and detailed recording of the findings at laparotomy will suffice to map out the tumor satisfactorily, it is often useful to insert radiopaque metal clips around its periphery for further reference. Figure 3 is a roentgenogram of another patient with a carcinoma of the bladder more extensive than that of the previous patient. One can see metal clips outlining the limits of the tumor as well as an extra clip at the site of an enlarged iliac node. This patient was also treated with cobalt^{60} irradiation. The radiation distribution is shown in figure 4. One can see that all of the tumor is enclosed within the area of high radiation. The skin dose is quite unimportant; the critical organ here is the rectum, which gets about 70 to 80 per cent of the tumor dose, but this is still tolerable.

The point we are trying to clarify is whether better results can be obtained by availing ourselves of what is routine information for a surgeon—to have an opportunity to see that cancer before we treat it.

Exploratory laparotomy performed solely to obtain information may be of great value in treating many patients having lesions of other sites when a radical radiation treatment is contemplated.

The mortality rate for this type of operation should be no higher than that for uncomplicated appendicitis, provided patients in poor general condition are excluded. If they are not well enough to tolerate this simple procedure, they would not be candidates for even a moderately intensive radiation treatment. Whether the morbidity, inconvenience, and expense of an

FIG. 2. Diagram showing a side view of the same tumor shown in figure 1.

exploratory laparotomy is justified by the information one expects to obtain is a matter for careful consideration. It obviously should not be recommended routinely; in many cases it would be quite unnecessary and wasteful. We believe, though, that its indications are broader than are commonly held, and that in selected patients there should be no hesitation in going ahead with it.

At times the decision to go in and have a look can be doubly justified by the presence of an unrelated pathologic condition requiring excision: gall bladder stones, uterine fibroids, and others.

In the next 2 cases the diagnosis was known but the exact location of the disease was not. A 21-year-old woman with Hodgkin's disease had previously had radiation treatment to the supraclavicular and axillary areas as well as the mediastinum. At the time we saw her she was very ill with profound anorexia, weakness, a 20-lb. weight loss, and itching. No nodes, however, were palpable in the peripheral lymphatic regions. Palpation of the abdomen was equivocal; there seemed to be a feeling of resistance here and there, but nothing of which we could be sure. At laparotomy we found tremendous involvement of the para-aortic nodes (fig. 5). In addition, there were nodes at the porta hepatica and along the mesentery of the descending colon. This latter area of involvement is rather rare and it is doubtful whether we would have thought of directing the radiation beam there. After a rather prolonged course of treatment the patient was rendered asymptomatic and is now leading a normal life.

A middle-aged woman, suffering from Hodgkin's disease which had been held in abeyance for some 7 years by treating the areas of tumor involvement as they became evident, was having mild systemic symptoms without any detectable nodes. She had been troubled at times by cholecystitis and had gallstones, shown by x-ray examination. We decided there were clear indications to have the gall bladder removed. At laparotomy we found a group of enlarged lymph nodes around the celiac artery (fig. 6), an area which is almost impossible to palpate through

FIG. 3. Roentgenogram of a patient with an extensive carcinoma of the bladder

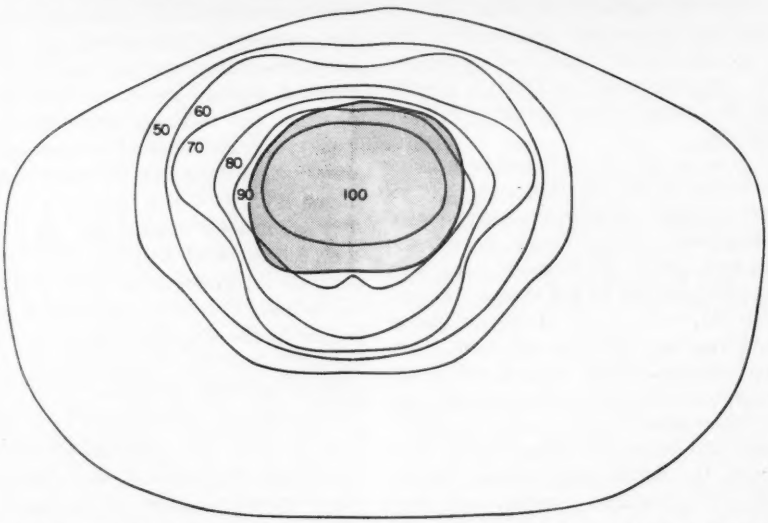

FIG. 4. Isodose lines showing distribution of the radiation given to the patient with extensive carcinoma of the bladder (fig. 3).

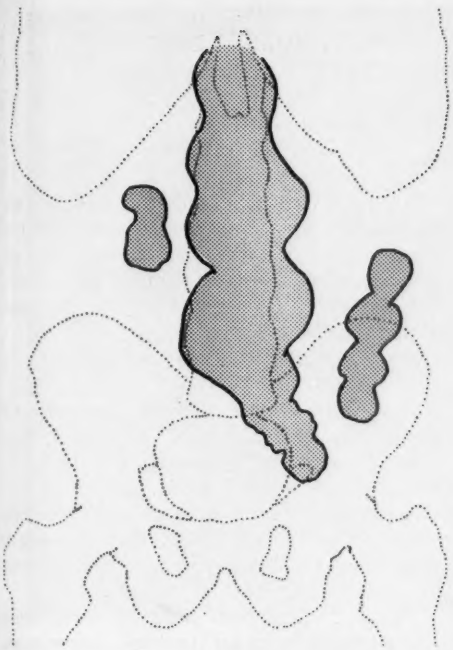

FIG. 5. Diagram showing tremendous involvement of the para-aortic nodes in a patient with Hodgkin's disease.

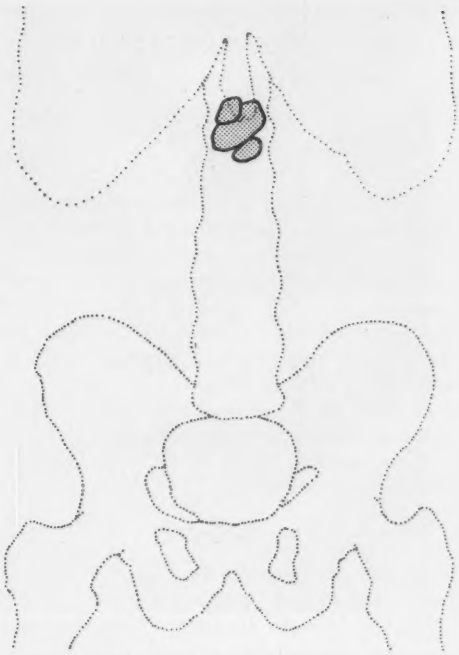

FIG. 6. Diagram showing a group of enlarged lymph nodes around the celiac artery in a patient with Hodgkin's disease.

the abdominal wall. After treating those nodes the patient once again went into a remission.

Frequently we find a patient who at operation is found to have an unresectable malignant tumor of a kind that is still treatable by radiation therapy. Although in most of these patients the radiation treatment would have only a palliative effect, occasionally cure can be attempted. Again, however, it is important for the radiotherapist to know the exact extent and location of the tumor if geographic misses are to be avoided. It is unfortunate that this information is rarely available, although there was a good occasion to record it. All too often operative reports give a detailed description of the kind of silk used to close the skin and many other insignificant technicalities, whereas only a few inadequate words are devoted to describing the lesion. If a radiotherapist is to make a guess-free treatment plan for a tumor he will never see, he should be provided with precise information with measurements of the tumor in three dimensions, as well as a description of its relation to neighboring organs and skeletal landmarks. Modern technical advances in radiotherapy and even clinical applications of new and exciting radiobiologic concepts are quite valueless if part of the tumor is left outside the beam of radiation.

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EXPERIMENTAL REPLACEMENT OF THE COMMON BILE DUCT WITH TEFLON GRAFT*

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Surgical repair of a strictured common bile duct is still a complex technical problem. The results of previous investigators searching for a suitable bile duct prosthesis have been disappointing.[1, 2, 9] Early failures were caused by leakage or disruption of the anastomotic line.[5, 12] Late failures were caused by partial or complete obstruction of the prosthesis.[1, 4, 7, 8]

Teflon grafts have been used successfully in other fields of surgery. This success stimulated us to investigate the applicability of Teflon in bile duct reconstruction.

METHOD

Fifteen mongrel dogs, weighing 17 to 24 kg., were anesthetized with intravenous sodium Pentothol. A right subcostal incision was employed to gain adequate exposure of the common bile duct. Dissection of the bile duct was carefully limited to that part necessary for excision and anastomosis. A segment of duct varying from 2 to 4 cm. in length and distal to the cystic duct was excised. The sphincter of Oddi was preserved in all cases. The gall bladder was not removed. A free Teflon graft $\frac{3}{16}$ inch in diameter and varying from 2 to 5 cm. in length was anastomosed to the severed ends of the common bile duct, without intraluminal splints. The anastomoses were performed with a continuous over-and-over suture of 5-0 arterial silk. A portion of gastrohepatic omentum was sutured loosely over the grafted area. The operative area was drained with a Penrose drain exteriorized through a stab wound in the midline. In the immediate postoperative period the dogs received intravenous fluids, then diet as tolerated. Penicillin and streptomycin were administrated once daily for 3 to 5 days. Urine specimens were collected at intervals and examined for bilirubin and urobilinogen content. The dogs were weighed preoperatively and before autopsy.

RESULTS

Of 15 dogs, 13 dogs survived replacement of the common bile duct with Teflon graft. One death occurred during surgery, and 1 animal died 12 hours postoperatively. Among 13 surviving dogs, 11 have been sacrificed at intervals varying from 42 to 365 days. The remaining 2 dogs, which are alive and well, will be sacrificed at a later date.

The 11 animals that were sacrificed were clinically well, with good appetites; their wounds were well healed without excessive drainage. They showed no evidence of jaundice and their stools were not acholic. Urine studies disclosed an immediate bilirubin increase and urobilinogen decrease; within 96 hours these had returned to normal preoperative levels. Eight of these animals gained from 0.5 to 2 kg. of weight; 3 animals lost 1 to 2 kg. of weight.

Gross autopsy findings in the 8 animals that gained weight were similar. There were no adhesions or evidence of bile peritonitis. The common bile duct, including the grafted area, was covered by a firm, smooth, yellowish white tissue (figs. 1 and 2). On section, this covering was found to encircle the entire graft and appeared to be continuous with the wall of the common bile duct above and below the anastomoses (figs. 3 and 4). The suture lines were smooth, without gross evidence of foreign body granulomas (fig. 5). There were no bile concretions or calculi within the graft or bile duct, and no gross evidence of epithelization within the Teflon graft. The grafted segment showed no evidence of shortening (fig. 6). The bile duct proximal and distal to the anastomoses was not dilated, nor was the wall thickened. The gall bladder was not enlarged, and no liver abscesses

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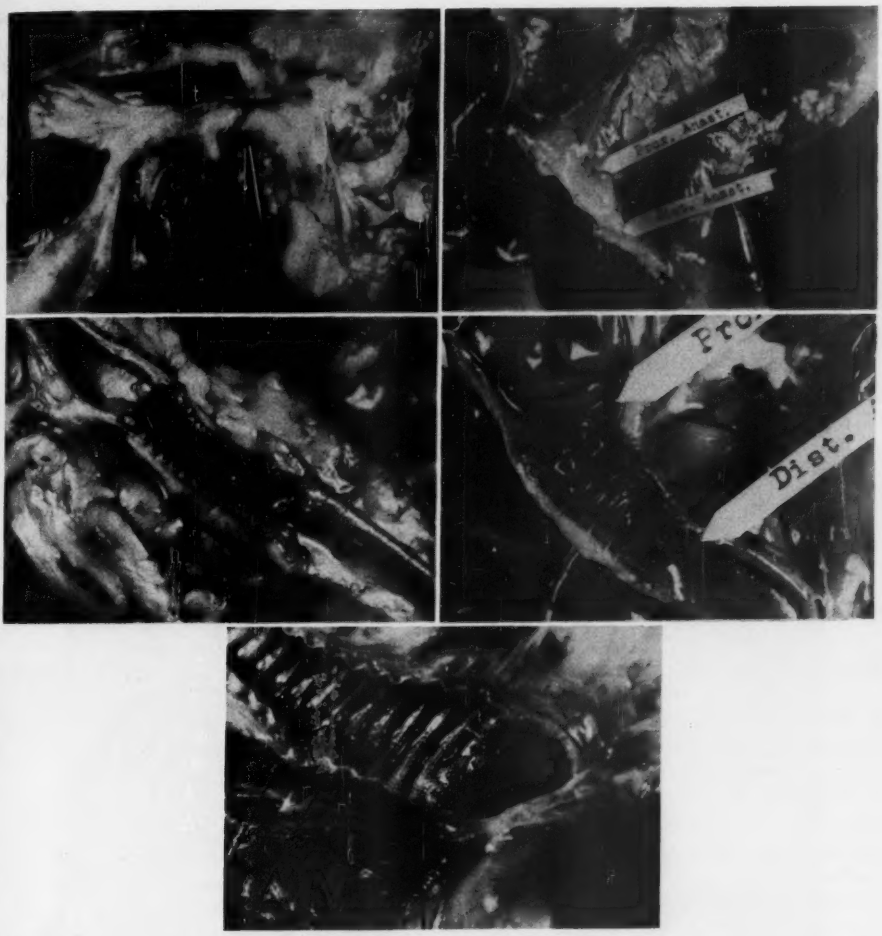


FIGS. 1 TO 5

FIG. 1 (*upper left*). A 42-day survivor; growth of tissue over Teflon graft and common bile duct. Gall bladder at lower left corner. Instrument points to position of Teflon graft.

FIG. 2 (*upper right*). A 365-day survivor; growth of tissue over Teflon graft and common bile duct. Note lack of adhesions.

FIG. 3 (*middle left*). A 42-day survivor; longitudinal section. Teflon graft surrounded by newly formed tissue. Note growth of tissue into the folds of the crimped Teflon graft.

FIG. 4 (*middle right*). A 365-day survivor; longitudinal section. The covering that surrounds the Teflon graft is continuous with the common bile duct proximal and distal to the anastomoses.

FIG. 5 (*bottom*). A 300-day survivor; longitudinal section. Proximal anastomosis. Note smooth, intact suture line without evidence of foreign body granulomas.

were encountered. The duodenum and stomach were bile-stained and showed no ulcer formation.

Microscopically, the new channel surrounding the graft was composed of fibrous connective tissue with collagenous fibers and fatty tissue. Within this fibrous tissue an inflammatory reaction was found which decreased in intensity as the survival time increased. The fibrous tissue had grown into the folds of the crimped Teflon graft (fig. 7). There was no epithelization within the Teflon graft, or within the newly formed channel. For example, in the 42-day survivor (fig. 8), focal chronic inflammation was noted within the dense, fibrous connective tissue.

FIG. 6. A 300-day survivor; longitudinal section. Note that there are no bile calculi or evidence of epithelization within the Teflon graft. There is no shortening or contraction of the graft.

Blood vessels were still present, and the channel had not undergone complete hyalinization. In the 365-day survivor (fig. 9), there was a dense fibrous connective tissue, fatty tissue and an absence of inflammatory cells.

Among the 11 animals sacrificed, 3 had lost weight. Two of these dogs, at 60 and 72 days, showed massive adhesions between the stomach, duodenum, liver and biliary tract. In each specimen the Teflon graft was found to be lying free within the newly formed channel, disrupted from its anastomoses and displaced towards the duodenum (fig. 10). There were no concretions, and no epithelial lining within the Teflon graft or within the newly formed channel. The proximal and distal anastomotic areas were partially stenosed, with dilation of the duct proximally (fig. 11). The gall bladder was distended and its wall was thickened. The liver showed no gross evidence of abscess formation. The duodenum and stomach appeared normal, with no evidence of ulcer formation. The 3rd dog (124 days), had two unsuspected anomalous bile ducts. These ducts arose from the left lobe of the liver and entered the common bile duct distal to the distal anastomosis, approximately 2 cm. from the duodenum. This dog also had a large subdiaphragmatic hematoma.

In the 3 dogs with postoperative stricture, the microscopic findings were similar. The strictured channel showed a marked increase of fibrous connective tissue. Within this fibrous tissue a marked inflammatory reaction was noted (fig. 12). Multiple abscesses with bacterial colonization were seen. There was no epithelization. There was an acute cholangitis of the duct, and in the liver (fig. 13) there was an acute cholangiolitis with abscess formation.

DISCUSSION

"The common purpose of any foreign object is to serve as a support or scaffolding while healing of adjacent or anastomosed tissue is occurring."[11] Teflon graft in our experimental study of replacement of the common bile duct served as a good support and scaffolding, and new channels were formed in all cases.

The problem of leakage or disruption of the anastomotic lines was not evident in our successful series of dogs. The use of continuous over-

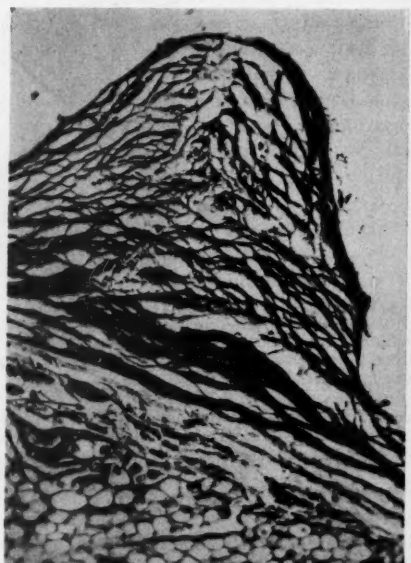
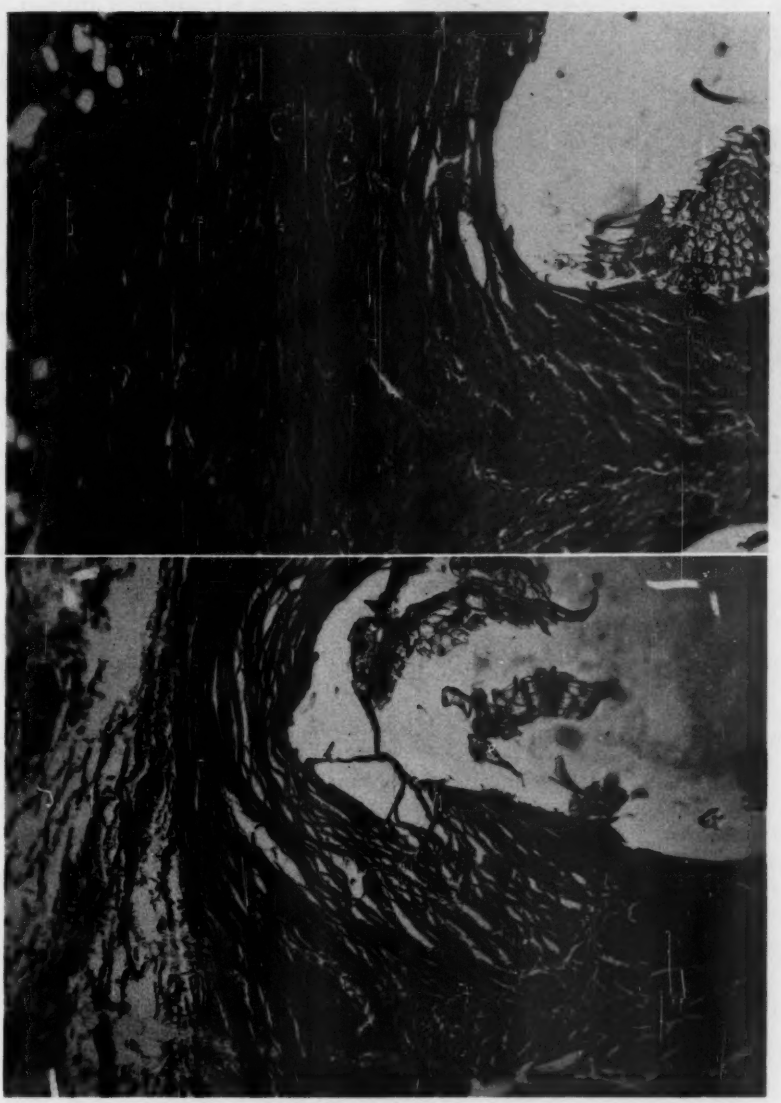


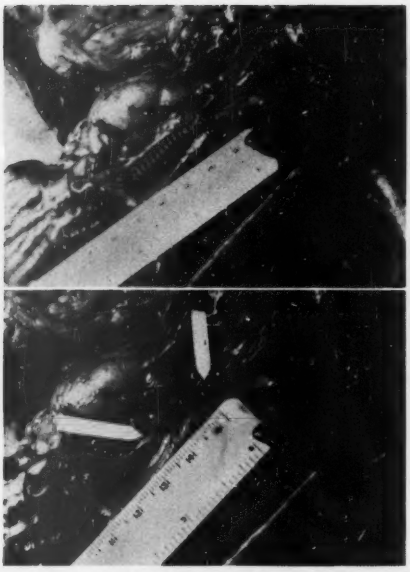
FIG. 7. A 300-day survivor; section through wall of channel. Note growth of fibrous collagenous tissue into a fold of the crimped Teflon graft and absence of inflammatory cells.



FIGS. 8 AND 9

FIG. 8 (*top*). A 42-day survivor; section through wall of newly formed channel. Early hyalinization with focal chronic inflammation within dense collagenous fibrous tissue. Note blood vessels. Fatty tissue at left and fragment of Teflon graft at right of picture.

FIG. 9 (*bottom*). A 365-day survivor; section through wall of channel. Note fatty tissue and dense collagenous fibrous tissue with absence of inflammatory cells.



FIGS. 10 AND 11

FIG. 10 (*top*). A 72-day survivor with partial stricture. Teflon graft lying free within the newly formed channel and displaced towards the duodenum. Common bile duct dilated proximally.

FIG. 11 (*bottom*). A 72-day survivor with partial stricture (Teflon graft removed). *Arrows* indicate areas of stenosis at the anastomotic lines. Note longitudinal shortening of the channel (original, 3½ cm.) and dilated common bile duct proximally.

and-over nonabsorbable sutures enabled a secure closure with minimal or no bile leakage. An omental flap, placed over the graft,[12] not only aided the development of a new channel, but also enhanced the blood supply to the critical lines of anastomoses. Pressure necrosis, which occurs with metal prostheses, is less likely to occur with Teflon.

Teflon is a fluorocarbon plastic. It is flexible, soft and nonbrittle, and is easy to handle and suture without collapsing or kinking. Is property of low tissue reactivity and zero moisture absorption favors a more rapid healing of the implant. Teflon is not degraded by acids, alkalis, organic chemicals or fluids. It retains its original strength as a matrix for the ingrowth of fibrous tissue.

We believe that the majority of our grafts were successful because of the lack of excessive inflammatory reaction about the anastomotic lines. The immediate cause of inflammation during such a procedure is irritation from bile.[6] In the 3 dogs with stricture formation, bile leakage at the anastomotic lines caused a marked inflammatory reaction with increased fibrous tissue in the wall of the channel, which was unsupported because the Teflon graft had become disrupted from its anastomoses and was displaced distally. Microscopic findings in these 3 dogs included large areas of inflammatory tissue surrounding clumps of bacteria. Stricture occurred after the disrupted grafts were displaced distally. Insufficient blood supply at the anastomotic lines was the most likely cause for this disruption.

The persistence of inflammatory cells in 3 dogs with unsupported and strictured channels was caused by the sustained irritation of septic bile. Santos and co-workers[10] consistently found on random sampling that dog bile contains various bacteria. Among 11 dogs sacrificed, there was no evidence of obstruction of the prosthesis from bile concretions, and no collapse or shortening of the graft. This success may be attributed to the properties of Teflon, which is not only inert, firm and nondeteriorating, but also sufficiently "nonwettable" to resist calculus formation.

One operative death was due to an overdose of anesthetic agent. One animal died 12 hours postoperatively; the cause of death could not be determined at autopsy. However, this death might be explained by excessive spillage of bile during the operative procedure,[3, 6, 10] for this dog had a very small bile duct and the anastomoses were accomplished with difficulty.

SUMMARY

Of our 15 attempts in dogs to reconstruct the common bile duct with the use of Teflon grafts, 10 can be considered successful. Eleven dogs have been sacrificed at intervals of 42 to 365 days, and at autopsy 8 dogs disclosed no evidence of early or late complications. Two animals are still living and are clinically well beyond 465 days.

The use of Teflon graft in reconstructive common bile duct surgery merits further consideration.

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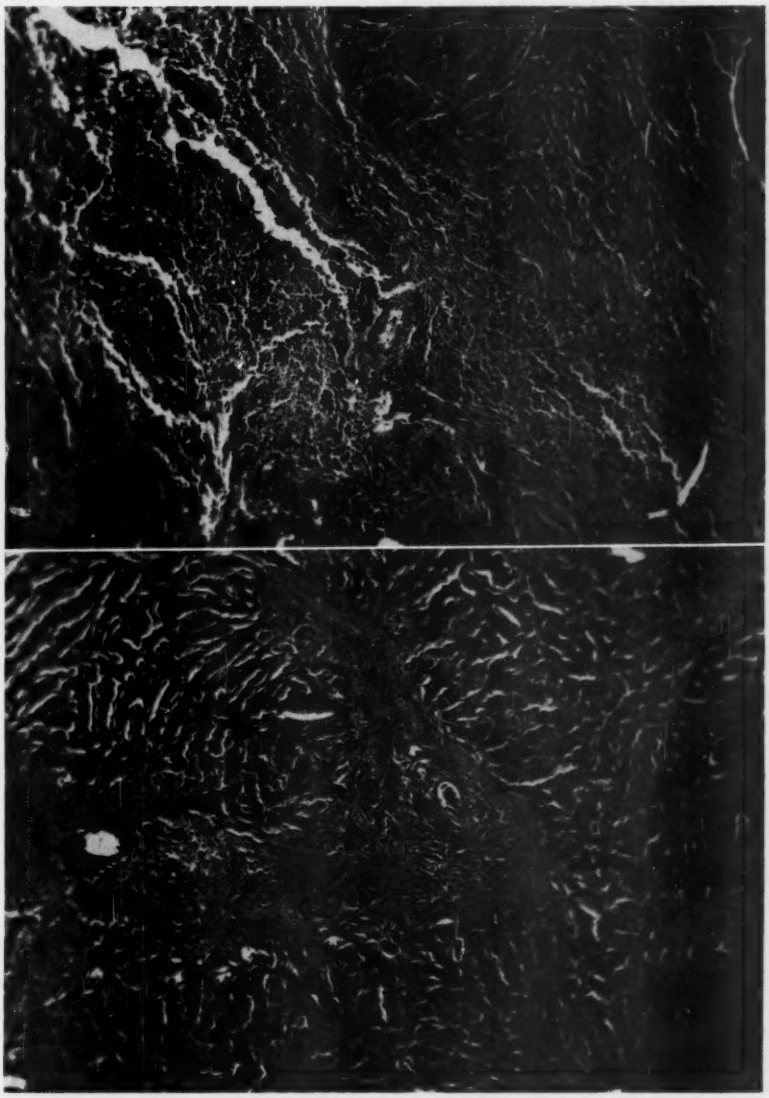

FIGS. 12 AND 13

FIG. 12 (*top*). A 72-day survivor with partial stricture; section through wall of newly formed channel. Marked acute inflammatory reaction within dense fibrous tissue. Necrosis and abscess formation.

FIG. 13 (*bottom*). A 72-day survivor with partial stricture; section of liver showing acute cholangiolitis with abscess formation.

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CRITERIA FOR SURGICAL CARE IN HIATAL HERNIA

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Because criteria should be established for the surgical therapy of hiatal hernia, a classification was devised some 8 years ago to use as a guide. This was published in *The American Surgeon* 3 years ago.[2] Sixty-five consecutive patients with hiatal hernia (table 1) have been evaluated and this clinical-pathologic classification, outlined below, was used to categorize each case.

Clinical-Pathologic Classification

1. Asymptomatic
   a. Small hernia
   b. Large hernia
2. Obstruction
   a. Esophagitis
   b. Ulceration
   c. Stenosis
3. Bleeding
   a. Incarceration
   b. Ulceration
   c. Gangrene
4. Pain
   a. Retrosternal
   b. Epigastric
   c. Referred

This report is an analysis of my experience with this approach and conclusions drawn as to proper criteria for selection of therapy.

The classification is unique in that it combines the important considerations of size of hernia, pathology resulting from the hernia, and symptomatology. Despite this seemingly all-inclusive approach, this classification roughly parallels the indications for surgical care of peptic ulcer, aside from the consideration of size.

The cardinal symptoms of hiatal hernia are comparable to those of peptic ulcer; *i.e.*, obstruction, bleeding and pain. In addition, certain large asymptomatic hiatal hernias deserve special consideration. These prime complaints are designated and the number of patients falling into each category are enumerated. The predominance of the symptom of pain is most significant. Pain was the prime complaint in 48 (73 per cent) of the total 65 cases; 3 patients were asymptomatic; and in 7 patients each obstruction and bleeding were the cardinal symptoms.

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Asymptomatic. The fact that a patient without symptoms may be a candidate for operative repair of the hernia emphasizes the importance of size of the hernia.

No patient was referred for evaluation of a small asymptomatic hernia. A small hernia is found so often at the time of roentgen study of the upper gastrointestinal tract, that its frequency borders on routine. Van Hazel and Jensik[5] termed it the second most common finding in roentgen studies of the upper gastrointestinal tract.

A large asymptomatic hernia presents a different problem because of the likelihood of further trouble. Blades and Hall[3] reported that the consequence of neglecting large hiatal hernias was a 50 per cent incidence of serious complication. They therefore advocated rpeair when feasible. In this series 3 patients had large asymptomatic hernias and 1 was operated on with a good result. The other 2 patients were advanced in age and their general condition was so poor that the hazard of surgery mitigated against operation.

Obstruction. Dysphagia may be present in varying degrees depending upon the severity of esophageal pathology, ranging from minimal esophagitis through stenosis. Dysphagia was the prime complaint in 7 patients and occurred as either the main or secondary complaint in 30 patients (46 per cent).

Esophagoscopy is most useful to determine the presence and type of esophageal pathology and to rule out carcinoma. Of the 30 patients complaining of dysphagia, the following types of pathology were discovered: esophagitis, 19; ulceration, 6; and stenosis, 5.

There is good correlation between the triad of dysphagia, retrosternal pain and esophagitis. The symptom of a "sticking" sensation associated with minimal edema and reddening of the esophageal lining may progress through advanced esophagitis with severe pain and dysphagia to

TABLE 1

Hiatal hernia: 65 cases

Sex	Males 36, females 29
Average age	55 years
Age range	28–82 years
Duration of symptoms	Average, 27 months
Type of hernia	Paraesophageal, 15%; sliding 85%

complete obstruction caused by stenosis. Sprafka and co-workers[6] followed 135 patients with hiatal hernia for 1 to 5 years and reported an increase in the size of the hernia in 13 per cent of the cases and a progression of lower esophageal pathology to ulcer, esophagitis and stricture in 20 per cent.

Because of this known progression of pathology, a patient with established esophagitis should respond promptly and adequately to a medical regimen or submit to surgery. The progression to a stenotic state must be avoided because of the radical and largely unsatisfactory surgical procedures available for the treatment of stenosis. Thomas and Merendino[8] have recently reported success with jejunal interposition, but a procedure of this magnitude may be avoided by proper treatment to prevent the development of stenosis.

Bleeding. In 7 cases, bleeding was the primary complaint and a secondary symptom in an additional 2 patients. The origins of bleeding were: incarceration, 4 cases; ulceration, 5; and gangrene, 1.

Sweet[7] has re-emphasized that a chronic secondary anemia is frequently found with hiatal hernia. This is particularly true if the herniated gastric mass is fixed and under torsion, whereas severe or massive bleeding is most commonly associated with gastric ulcers lying within the herniated stomach. In this series there were 5 instances of massive hemorrhage due to gastric ulceration within the hernia. Although Feldman and Myers[4] have demonstrated statistically that gastric ulcers occurring in hiatal hernia are no more common (4 per cent) than in the general population, when a bleeding ulcer is found in a hernia it poses a more serious problem.

One patient had a large part of the stomach (one-third) incarcerated in the hernia. This became gangrenous with massive bleeding. This is an extremely rare complication of hiatal hernia and demands immediate blood replacement, gastric resection and repair of the hernia. This patient had a good result.

Some of the more salient points of importance in the management of patients with massive bleeding are illustrated in the following case report.

CASE REPORT

R. K., a 55-year-old housewife, was admitted to the hospital with a long history of dysphagia thought to be due to an esophageal diverticulum. She had a 2-week history of epigastric pain, tarry stools and weakness. Adequate medical treatment was begun but melena persisted, and hemoglobin values decreased from 75 per cent on admission to 63 per cent 3 days later despite two blood transfusions. Shock ensued, and after correction by blood replacement a roentgen study of the esophagus and stomach was done and an active ulcer lying within a large, sliding hiatal hernia was demonstrated. The patient had had eight abdominal operations during the previous year for treatment of a perforated sigmoid diverticulum with peritonitis and subsequent intra-abdominal abscesses. A transthoracic approach gave easy access to the hernia and the ulcer lying along the lesser curve of the herniated cardia. Good recovery and results were obtained.

This patient presented each of the cardinal symptoms of hiatal hernia, but the main problem was that of massive bleeding and shock. Early and adequate blood replacement demonstrated its effectiveness in combating shock without contributing to prolonged bleeding. The value of early roentgen studies to aid in diagnosis was most dramatic since, in this instance, had no roentgen studies been made and had operation been undertaken for bleeding duodenal or gastric ulcer *via* the abdominal approach, the presence of massive intra-abdominal adhesions would have made exposure and diagnosis forbidding. The successful hernia repair and ulcer excision would have been almost impossible had not the transthoracic route been employed.

Pain. An understanding of pain is most important in both the diagnosis and evaluation of patients with hiatal hernia. Pain was the cardinal symptom in 73 per cent of the cases; the symptom of pain was present in every symptomatic case (62) and absent only in the 3 asymptomatic patients. Thus, it can be said that if the hernia is symptomatic, one of the complaints will be pain. Similarly, a knowledge of the various pain patterns aids immeasurably in evaluating the effects of the disease. I have

found it useful to assign pain to one of three "patterns." For this group, 62 cases, the pain pattern was retrosternal in 46 patients, epigastric in 51, and, referred in 5.

Retrosternal "burning" pain of esophagitis, due to reflux of gastric contents past the gastroesophageal juncture, occurs most commonly as a result of the effect of gravity in the forward bending or reclining position, or with increased intragastric pressure associated with exertion or obesity.

Epigastric "full, steady" pain behind the xiphoid or beneath the left costal margin or precordium generally follows meals.

Referred "sharp, stabbing" pain over the precordium, radiating to the neck, shoulders and arms, also occurs after meals or exertion. This gives rise to the serious question as to whether the pain is due to the hernia or the heart. There were 7 patients with coronary artery disease in this series.

A recognition of these pain patterns will allow an understanding of the complete clinical picture. In the 65 cases reviewed, isolated pain patterns were the exception. Only 4 patients had retrosternal and epigastric pain alone, and each of the 5 patients with referred pain had associated retrosternal and epigastric pains.

SELECTION OF PATIENTS, MANAGEMENT AND RESULTS

The main indications for selection of patients for surgical or medical management are tabulated in table 2.

TABLE 2

Indications for type of treatment

Indication	No. of Cases
Surgical	
Uncontrolled pain	10
Pain and obstruction	19
Pain and bleeding	6
Large asymptomatic hernia	3
Total	38
Medical	
Controlled pain	15
Pain and obstruction	10
Pain and bleeding	2
Total	27

TABLE 3

Surgical procedures

Type	Frequency
Transthoracic repair (Allison technique)	17
Transthoracic repair, gastroenterostomy, vagotomy	3
Transthoracic repair and wedge gastrectomy	4
Transthoracic repair and esophagectomy	1
Transabdominal repair	1
Transabdominal repair and cholecystectomy	3
Transabdominal repair and gastrectomy	3
Esophageal dilation	3
Pyloroplasty	2
Transthoracic vagotomy	1
Total	38

The words "controlled" and "uncontrolled" are the keys to the selection of medical or surgical care in hiatal hernia just as in peptic ulcer. Absolute indications for surgery are continued massive bleeding, stenosis, huge hernia, carcinoma in a hernia and gangrene of an incarcerated gastric mass. With the exceptions of these absolute indications, the best criterion for successful management is the type of response to adequate medical management and to weight reduction. The large number (27) of patients whose symptoms were severe enough to warrant thorough evaluation, yet who responded adequately to a medical regimen, is testimony to the effectiveness of medical care alone in many instances.

SURGICAL PROCEDURES

Some 38 procedures were carried out on the 38 patients selected for some type of surgical management. The Allison technique was used in most instances of operative repair (table 3).

The ideal in surgical treatment is the correction of both the hernia and associated pathology. This ideal was achieved in 31 of the 38 cases, but in 7 instances the ultimate in surgical care was either not attempted or failure resulted: 1 patient with carcinoma of the esophagus in a with stenosis were treated by dilation alone;

and 1 patient was treated by transthoracic vagectomy alone because of a "frozen" fibrotic esophagus due to long standing inflammation. The 2 patients treated by pyloroplasty alone were medical failures, but their general condition seemed to forbid a surgical procedure more drastic than pyloroplasty. Both had excellent symptomatic relief.

In the 31 patients who received definitive surgical procedures, the preferred approach was transthoracic except when coexisting abdominal pathology dictated simultaneous correction. The transthoracic approach was preferred (25 patients) because anatomic correction of the defect was more easily obtained.

SUMMARY

Sixty-five consecutive patients were evaluated because of hiatal hernia. These cases have been reviewed to determine the usefulness of categorizing patients according to a devised clinical-pathologic classification and to determine proper criteria for surgical care.

Large asymptomatic hernias should be repaired because of the serious complications of long standing neglect, provided the patient is in satisfactory condition. The uncontrolled complications of hiatal hernia (continued bleeding, carcinoma, stenosis and gangrene) demand surgical care and form the absolute indications for surgery. For most patients with hiatal hernia, however, an adequate course of medical management should be carried out, surgical measures being reserved for those with uncontrolled symptoms or pathology.

Pain, bleeding and obstruction form the three cardinal symptoms that reflect the corresponding underlying pathologic alterations due to hiatal hernia, esophagitis, ulceration and stenosis.

If dysphagia caused by esophagitis or ulceration does not respond rather promptly to a medical regimen, surgery should be carried out to prevent stenosis.

If bleeding persists or recurs, operation is indicated in, roughly, the same circumstances as in bleeding from peptic ulcer.

If pain is disabling and intractable to medical care, repair of the hernia is indicated, not only for relief but for prevention of progression of the underlying pathology.

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PRACTICAL PROBLEMS IN THE TREATMENT OF HEPATIC TRAUMA

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The mechanistic age in which modern man lives is a traumatic one; hence, the abdominal viscera are often injured by accidents, occurring at home, on the highway, at work or at play. The liver, being the largest and heaviest organ of the body, often receives both penetrative and blunt impacts. Because it performs so many vital functions, any traumatic insult, of necessity, produces severe constitutional reactions. Unless these pathologic dysfunctions are corrected, death may follow.

TYPE OF INJURY

Hepatic wounds are classified as penetrative, lacerative and subcapsular.

Penetrative wounds perforate Glisson's capsule and damage the contained parenchymatous structures. For example, a bullet of small caliber may pass through the liver without causing much damage; or it may sever some of the larger blood vessels and bile ducts, thus producing hemorrhage and extravasation of bile. High velocity missiles not only produce a perforative tract or channel, but also kinetic energy often injures tissue surrounding the sinus tract. Blasts from a shotgun have been known to avulse large pieces of liver without causing much intra-abdominal bleeding, whereas even trivial injury has produced exsanguinating hemorrhages. Foreign objects, such as pieces of clothing, cellucotton, gunpowder, bullets, metal objects, or fragments of bone, are often embedded in the liver substance. These foreign materials are not well tolerated, and should be removed.

Stab wounds, through the chest or the abdominal wall, which injure the liver, require special consideration. The external opening of the wound, though small, often results in considerable damage, by laceration, to the underlying liver substance. One should not be lulled into a sense of false security because of the smallness of the wound.

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Fragmentation, laceration, and rupture of the liver usually occur when the abdomen receives a sudden compressive impact. The blunt force often ruptures Glisson's capsule, compressing and macerating the hapatic substance. If the laceration involves the hilum of the liver, exsanguinating hemorrhages may result. As a rule, however, lacerations involve the dome of the liver, producing large gaping wounds from which both blood and bile escape into the peritoneal cavity.

In subcapsular injuries to the liver, Glisson's capsule remains intact, whereas the parenchymatous tissues become torn and disorganized. This permits blood and bile to extravasate into the liver substance. As this intrahepatic abscess increases in tension it tends to burst or tear, producing large hematomas which may rupture at Glisson's capsule, liberating both blood and bile. These "delayed ruptures" of the liver often prove fatal.

Sometimes subcapsular hematomas do not rupture Glisson's capsule but, instead, erode into the larger bile ducts and evacuate their contents, *via* the ducts, into the duodenum. This syndrome is called "hemobilia." Under such circumstances the surface of the liver is smooth and not traumatized, and there will be no free blood in the peritoneal cavity. Sparkman and Forgelman[2] described one such case, in which the hematoma was not discovered until it had produced a fatal exsanguinating melena.

As a result of his war experiences, Madding[1] believes that intrahepatic abscesses develop when a laceration of the liver is approximated by sutures without employing adequate drainage. It is imperative that drains be left *in situ* until the deeper portions of the liver have completely healed. We encountered this phenomenon in a patient in whom a large subphrenic hematoma had converted into an abscess because of the adherent diaphragm. The abscess cavity eroded into the liver, finally evacuating its contents, by way of the larger bile ducts, into the duodenum.

Because of its size and location, the right

lobe of the liver is injured more often than any of its other segments. In 829 cases of hepatic trauma incurred in the war, Madding found the right lobe involved in 86.9 per cent, the left in 13.1 per cent, and both lobes in 3.4 per cent. In 28 instances of liver trauma occurring in civilian practice, we found the right lobe involved in 92 per cent of the patients studied, the left lobe in 8 per cent, and both lobes in 4 per cent. Most injuries involved the dome and exterior surface of the right lobe. Sixty-four per cent of the patients had but a single laceration; 36 per cent showed multiple lacerations, the maximal number being 8. Whenever there were multiple lacerations they seemed to converge at one central point, and often resulted in avulsion or extrusion of small segments of the lobe.

In view of the fact that most injuries involved the entire abdomen, it is not surprising that other viscera often exhibited changes from severe impacts. Madding found that 59 per cent of 829 patients suffered from associated trauma to other organs. In our own experience, 56 per cent of the patients injured had multiple injuries to other organs.

TREATMENT

There is no place for conservatism in the treatment of hepatic injuries. Prompt, definitive surgery is the most effectual in reducing both mortality and morbidity rates. Preoperative treatment must be direct as well as energetic if shock is to be corrected. The patient must be prepared for immediate surgical exploration. To delay intensifies the shock and hence, reduces the opportunity for recovery.

Surgical therapy has several aims: It must (1) control hemorrhage and stop extravasation of bile; (2) remove damaged and nonviable portions of the liver; (3) drain the liver wound and the abdominal cavity; (4) prevent infection; and (5) recognize and correct traumatic damages to other viscera.

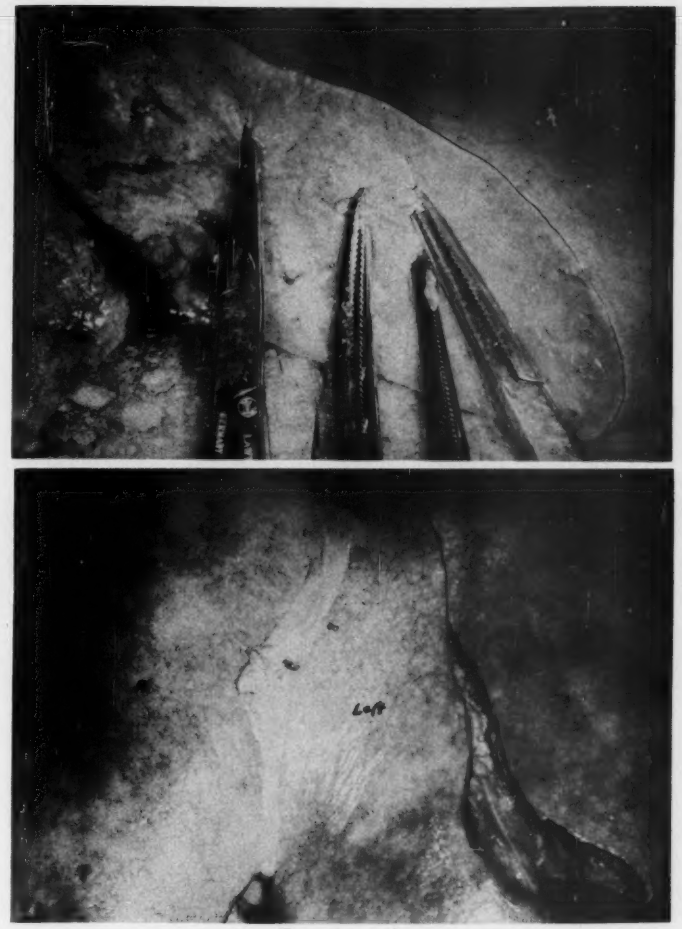
Control of hemorrhage requires complete visualization of the hepatic wound so that each major group of blood vessels and bile ducts may be occluded by direct application of suture ligatures. This cannot be accomplished until the abdomen has been evacuated of all loose fragments of liver and all blood clots. The "cupped hand" makes an excellent instrument with which to remove the solid and semisolid debris. After this has been accomplished the abdominal cavity may be irrigated with a warm saline solution, and aspirated. This maneuver, contrary to some opinion, does not increase hepatic bleeding. In 90 per cent of all patients, hemorrhage has stopped by the time the abdomen is opened. As a general rule, unless the laceration extends into the hilum of the liver, active bleeding will seldom be encountered. Temporary pressure with a hot gauze pack may prove helpful in controlling any venous or arterial oozing; and temporary compression of the hepatoduodenal ligament, either by digital compression or by means of a bulldog clamp, often minimizes troublesome bleeding so that direct ligation of the injured vessels can be accomplished.

Clinical and laboratory experience seems to indicate that hemostatic substances, such as Gelfoam, Oxycel, or gauze impregnated with fibrin, are of little value in the treatment of liver wounds. The liver seems to reject these foreign substances and in almost every instance expels them from the damaged area. This pnenomenon has been substantiated by laboratory studies with dogs that were laparotomized and their livers injured by either tearing, lacerating, crushing, or penetrating with pellets from air rifles. These damaged areas were then treated with Gelfoam, Oxycel, Ivalon, or gauze. In many instances these agents were sutured in the liver defects in such a manner that we did not think they could escape.

These dogs were sacrificed in a 5- to 10-week period. In most instances the implanted material had been expelled from the liver substance and was found to be free within the peritoneal cavity. In those cases in which small fragments of Oxycel or Gelfoam were apparently accepted by the liver, microscopic examination indicated that there was a marked foreign body reaction, giant cell infiltration, fibrosis, and granulomatous formation.

In a group of control animals, identical trauma was produced, but in no instance was any of the foreign material sutured in the liver and no attempt was made to repair any damage to the liver itself. The bleeding was controlled by suture ligation. It was interesting that the traumatized zones of the liver healed spontaneously.

It was difficult to say in many instances, either from gross examination, or microscopically, where the exact area of trauma had been. When segments of the liver had been removed, no



FIGS. 1 AND 2

FIG. 1 (*top*). Crushing the bile ducts and blood vessels as they appear on the cut surface of the liver, preparatory to definitive suture ligation.

FIG. 2 (*bottom*). A deep laceration practically bisecting the midportion of the left lobe of the liver. The devitalized peripheral segment was removed.

attempt was made to restore its normal anatomic profile; yet at autopsy the surface was covered by peritoneum identical to the uninvolved lobes. From these experiences, we feel it unnecessary to reconstruct the liver profile. The only active maneuver should be to control bleeding, to remove the devitalized liver tissue and to supply adequate drainage.

DEBRIDEMENT

Surgeons have been rather reluctant to accept the principle of debridement in dealing with hepatic injury. Perhaps fear of hemorrhage has been the deterring factor. When a segment of liver has been deprived of its blood supply it assumes a pale yellow, ischemic discoloration. This devitalized portion of tissue should be removed, otherwise it will undergo necrosis and slough away. When shreds of hepatic tissue hang from the margin of the laceration these projecting segments should be excised. If one should encounter a central hematoma, or area in which the central segment of the liver seems to be pulpified or macerated, this cavity should be opened, the

necrotic tissue removed, and adequate drainage employed. Experiences in World War II indicated that debridement was responsible for reducing mortality and morbidity rates associated with hepatic injury.

DRAINAGE

There is universal agreement that all wounds of the liver should be drained externally, but there is considerable controversy concerning the method to be employed and the materials to be used. It is generally assumed that gauze furnishes a matrix upon which blood may clot; that it exerts pressure to minimize arterial and venous bleeding; and, in addition, functions as a drain.

In actual experience, however, gauze does not siphon away the blood and bile but, rather, acts as a dam, causing a pooling of material to form abscesses. Madding has pointed out that serious secondary hemorrhages often occur when gauze is removed. Similarly, objections can be raised to the employment of gauze, Gelfoam, and oxidized cellulose. These materials invite infection, form abscesses, and may provoke secondary hemorrhages.

Blood and bile can be more effectively drained by using large pieces of rubber sheeting, solid rubber tubes, or Penrose drains. The rubber drains do not adhere to tissue, do not produce secondary bleeding, but do provide excellent channels for drainage. Tubes should be placed into the depths of a wound, and not at the periphery, since wounds must be kept open until deeper segments heal; otherwise, peripheral healing may close over, forming intrahepatic abscess.

When draining the liver, it is well also to place drains into the subphrenic space, into the subhepatic area, and into the right cecal gutter, since these are areas where serum tends to pool and promote secondary abscess formation. Drains should be anchored so that they will not be displaced too early, thus allowing surface healing or diaphragmatic adhesions to close wound margins and invite sequestrated intrahepatic abscesses.

INFECTION

One of the main functions of the liver is to filter and destroy bacteria which arrive by way of portal vein. The bile ducts communicate with the intestinal tract and frequently contain transient bacteria. It is quite obvious, therefore, that the bloody menstruum and bile of liver damage provide excellent culture media for bacterial growth. It seems prudent, therefore, to employ antibiotics to prevent this complication. Wide spectrum antibiotics which reach high levels in the bile seem preferable.

SUMMARY

The liver, which is the largest organ of the body, is frequently injured by both blunt and penetrative forces, with at least 50 per cent of such patients suffering from damage to other organs as well. Treatment is strictly surgical and consists of: (1) control of bleeding, and bile drainage; (2) debridement of devitalized liver tissue; (3) drainage of liver wounds and abdominal cavity; (4) control of infection; and (5) correction of damage to other organs. Clinical and laboratory test experience would indicate that the use of such hemostatic substances as Oxycel, Gelfoam and gauze packs is contraindicated in management of liver wounds.

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ACUTE PANCREATITIS IN THE NEWBORN, IN INFANCY AND IN CHILDHOOD*

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In the older literature acute pancreatitis in childhood has usually been attributed to mumps,[22] although it has also been associated with scarlet fever, diphtheria, typhoid fever, influenza, otitis media and trauma. Despite this variety of etiologic agents, it is evidently a rare condition in infancy and childhood. Blumenstock and others[6] have recently reviewed the literature and stated there were 36 such cases reported, to which they added 4. A short time later Stickler and Yonemoto[51] also presented a review stating that there were 38 such cases, including the 4 reported by Blumenstock and co-workers,[6] and they presented an additional case. In reviewing these and other reports it is evident that there are considerably more cases than reported either by Blumenstock's group[6] or by Stickler and Yonemoto.[51] We have been able to collect reports containing 68 cases *in toto*, to which we are presently adding 6, thus bringing the total number of cases of acute pancreatitis in children to 74.

The cases reported in the literature which have been considered as occurring in childhood have ranged from the newborn to 16 years, the latter evidently arbitrarily set as the upper limit for this age group. We have used the same age span in collecting the present series of cases. Our own cases, as well as those previously reported, have been separated into various etiologic groups according to a previously designed system.[7] This has afforded us an opportunity to compare the frequency of various etiologic types in childhood and adult groups.

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CASE REPORTS

Case 1. This male infant was born at full term, by a footling-breech delivery. He was cyanotic and bruised, and an incision of the maternal cervix was necessary for delivery. Shortly after delivery the infant's temperature rose from 97.5° to 102.4°. He developed a bilateral pneumonitis, abdominal vein distention and infection of the umbilicus. When respiratory difficulty developed, intratracheal suction was performed and adrenaline was given intracardially. However, he died 3 days after delivery.

At autopsy, blood culture showed a bacteremia with *Escherichia coli*. An acute inflammatory process was found in the lungs, meninges and pancreas, and there was bilateral adrenal hemorrhage. Because of the evidence of bacteremia, this case was placed in the infectious group.

Case 2. The mother in this case, a 36-year old gravida II, para I, had been treated for brucellosis 5 years previously. Two years prior to the present pregnancy she had had a spontaneous abortion. On this admission she delivered a 6 weeks premature living male infant with an infected omphalocele. The baby took a few gasps and died about 1 minute after delivery.

Autopsy showed an extensive organizing peritonitis in which infection had gained entry into the peritoneum through the infected omphalocele. There was old calcification of the adrenals, but no evidence of histoplasmosis or other specific organism. The acute inflammatory process in the pancreas was evidently due to extension of the peritonitis; therefore this was also considered as belonging in the infectious group, with infection occurring during intrauterine life.

Case 3. This is the case of a 3-year-old baby girl whose chief symptoms on admission were weakness, anorexia, bloody stools and epistaxis. She had mongoloid features. Leukemia was discovered on hematologic work-up. Her condition went downhill rapidly and she died about 3 weeks after admission.

Autopsy revealed an acute lymphatic leukemia, and postmortem blood culture showed a bacteremia with *E. coli*. An acute inflammatory process was found to involve the lungs, liver,

spleen and adrenals, as well as the pancreas. This case, therefore, was also placed in the infectious group.

Case 4. This 6 weeks premature female infant exhibited cyanosis within approximately an hour after delivery. Despite oxygen therapy this cyanosis continued over the ensuing 24 hours, when the child died.

Autopsy revealed atelectasis, a patent ductus arteriosus and foramen ovale as would be expected in a premature infant, extensive urate deposits in the renal pelvis, and an acute interstitial inflammatory reaction of the pancreas. Because of the evidence of persistent anoxia and the extensive urate deposits this case was placed in the metabolic group.

Case 5. This male infant was also about 6 weeks premature, but he lived for about 8 weeks after delivery. During this time he failed to show normal weight gain, developed anorexia and diarrhea which could not be controlled, and eventually died.

Autopsy revealed marked fatty metamorphosis of the liver and massive urate deposits in the renal pelvis similar to those found in case 4. The pancreas showed an acute interstitial inflammatory reaction also similar to that seen in the preceding case. Because of the changes in the liver and the urate deposits in the kidney, this was also placed in the metabolic group.

Case 6. This male infant was delivered by cesarean section about 2 weeks before the expected date. He developed signs of intestinal obstruction within the first few hours after delivery, and a laparotomy revealed a volvulus of the small bowel, with shortening of the related mesentery. After resection of the volvulus, two areas of atresia and one of stenosis were identified in the distal ileum, and an ileotransverse anastomosis was performed, with inversion of the short portion of the jejunum. Five days later a perforation occurred at the site of the inversion, and about 4 cm. of necrotic jejunum had to be resected; 6 days later two fistulas occurred because of necrosis of the transverse colon and ileum at the enterocolic anastomosis, and about 10 cm. of necrotic ileum had to be resected and a new ileocolic anastomosis established. The infant subsequently developed acute gastric dilation although the intestinal passage appeared free; he became worse and died at the age of 15 days, despite therapy.

Autopsy revealed some bile stasis in the liver, a serofibrinous peritonitis, and extensive interstitial acute inflammatory reaction of the pancreas. Because of the repeated surgical procedures and the absence of any other specific etiology, this case was considered as traumatic, of the postoperative type.

DISCUSSION

With the exception of a case of acute pancreatitis in a newborn infant reported by Gruenwald[27] and a case of acute pancreatitis superimposed on a pre-existing fibrocystic disease of the pancreas reported by Claireaux,[10] all other cases in infancy and childhood heretofore reported have been among patients 8 months of age or older. On the other hand, certainly our case 2 and possibly cases 4 and 1 developed their acute pancreatitis during intrauterine life. Furthermore, 5 of the 6 patients herein reported died from their pancreatic disease during the first 8 weeks of life. Thus, of the 74 cases in the literature, 8 (about 11 per cent) occurred immediately or shortly after birth. It is evident, therefore, that acute pancreatitis can occur at any time from birth on, and that some cases may even occur during intrauterine life. In this regard it is pertinent that Opie[42] casually mentioned a fetal form due to congenital syphilis.

Although, according to Stickler and Yonemoto,[51] acute pancreatitis in infancy and childhood has occurred about twice as frequently in females as in males, in the present group 4 of the 6 infants were males. However, the present group is too small to significantly alter this sex difference.

In a previous publication[7] we have shown that cases of acute pancreatitis can be divided into the following etiologic groups: (1) infectious, (2) obstructive disease of the biliary and/or pancreatic ductal systems, (3) metabolic, toxic or chemical, (4) traumatic, (5) vascular, (6) allergic, and (7) idiopathic. We have separated the cases reported heretofore and the 6 cases in the present report according to this grouping, as shown in tables 1 to 6. Stickler and Yonemoto[51] have presented a similar separation, but the basic difference is that they have placed in the idiopathic group cases which we have considered as occurring on an infectious basis.

If one considers associated evidence of infection as having etiologic significance, then 10 of the cases (13.5 per cent) fall into the infectious group. It is evident from table 1 that most of the cases in this group were associated with acute tonsillitis or otitis media. Two of the cases in

TABLE 1

Pancreatitis associated with infectious disease

Author	No. of Cases	Infectious Disease
Sebening,[47] 1925	1	Acute tonsillitis
Dobbs,[17] 1935	1	Otitis media with streptococcal brain abscess and erythema nodosum
Boss,[8] 1955	1	Infectious hepatitis, recurrent
Gibson and Gibson,[26] 1956	1	Otitis media with perforation
Pender,[43] 1957	1	Acute tonsillitis due to β-hemolytic streptococcus
Stickler and Yonemoto,[51] 1958	1	Acute tonsillitis with chlortetracycline therapy and urticaria
Collins,[11] 1958	1	Bronchopneumonia following burns
Present report	1	Bacteremia with acute meningitis
	1	Generalized peritonitis
	1	Leukemia with bacteremia and thrombocytopenia
Total	10	

TABLE 2

Pancreatitis associated with pancreatic or biliary ductal obstruction

Author	No. of Cases	Cause and Type of Obstruction
Novis,[41] 1923	1	Ascariasis (pancreatic duct)
Gallie and Brown,[24] 1924	1	Ascariasis (pancreatic duct)
Herzog,[30] 1929	1	Ascariasis (pancreatic duct)
Jaeger,[32] 1943	1	Ascariasis (pancreatic duct)
Duncan,[20] 1948	1	Ascariasis (pancreatic duct)
Schmieden and Sebening,[46] 1927	1	Gallstone (ampulla of Vater)
Blumenstock and others,[6] 1957	1	Secondary perforation of common bile duct on first attack; two putty-like masses in common duct on sixth attack
Total	7	

our series showed evidence of a bacteremia and the third of a generalized peritonitis. It is not evident how many of the other cases were associated with bacteremia, although 1 case[17] showed a metastatic brain abscess.

Seven of the cases (9.5 per cent) gave evidence of obstruction of either the pancreatic or biliary ductal system (table 2). In 5 cases this was evidently due to the round worm Ascaris and in only 1 case to a gallstone in the ampulla of Vater. It is evident that pancreatitis due to this latter cause is indeed rare in infancy and childhood. The case reported by Blumenstock and others[6] was evidently idiopathic initially, but on exploration following the sixth attack, two putty-like masses were found in the common duct. With the first attack a secondary perforation of the common bile duct had been discovered on exploration, and this required suturing. The two inspissated masses may have resulted from secondary stenosis of the duct and thus may have caused the subsequent attacks of pancreatitis. However, this patient did not exhibit jaundice, so that obstruction was never complete.

There were 24 cases (32.4 per cent) which were placed in the group due to metabolic, toxic or chemical factors (table 3). By far the largest number of these were reported by Steiner and Tracy[50] as associated with diabetic coma. However, this form of pancreatitis is probably disappearing, as a result of the excellent progress in preventing coma from diabetes. Derangement of

TABLE 3

Pancreatitis associated with metabolic, toxic or chemical factors

Author	No. of Cases	Metabolic, Toxic or Chemical Condition
Anderson,[3] 1923	1	Cirrhosis of the liver
Steiner and Tracy,[50] 1943	14	Diabetic coma
Gruenwald,[27] 1950	1	Anoxia
Collins,[11] 1958	3	Kwashiorkor; all cases had bronchopneumonia, 1 with staphylococcal abscess of lung; 2 had diarrhea and 1 thrombocytopenia
Baar and Wolff,[5] 1957	2	On cortisone therapy
Marcaynska-Robowska,[38] 1957	1	On cortisone therapy
Present report	2	Associated with extensive urate deposits in the kidneys
Total	24	

TABLE 4

Pancreatitis associated with trauma

Author	No. of Cases	Associated Findings
Connolly,[12] 1911	1	Pseudocyst
Dobrotvorsky,[18] 1912	1	
Hagedorn,[28] 1913	1	
Dietrich,[16] 1914	1	Pseudocyst
Drennen,[19] 1922	1	Pseudocyst
Venable,[55] 1932	1	
Liedbert,[35] 1937	1	
Kastrup,[33] 1938	1	
Adams,[1] 1939	1	
Shallow and Wagner,[48] 1947	1	
Mithoefer,[40] 1951	1	Pseudocyst
Singer,[49] 1952	1	Associated with trichobezoar and pseudocyst
Lussky,[36] 1955	1	Pseudocyst
Levitzky and others,[34] 1956	1	Pseudocyst
Present report	1	Postoperative in case of multiple congenital atresias of ileum
Total	15	

protein metabolism may constitute an important etiology. The disease kwashiorkor is known to be due to a diet deficient in protein and is prevalent in underdeveloped areas; 3 of Collins' cases[11] fit into this group, although there was also evidence of superimposed infection. The 3 cases on cortisone therapy may also be associated with a derangement in protein metabolism, since this hormone is known to depress protein synthesis. The presence of extensive urate deposits in 2 of our cases may be due to excessive protein catalysis. This is an important consideration since it is well known that the pancreas has a high protein requirement because of its function of synthesizing and secreting digestive enzymes which are largely protein in composition. Anoxia of the newborn may also be considered a metabolic factor, as in Gruenwald's case[27] and in our case 4.

There were 15 cases (20.3 per cent) associated with abdominal trauma (table 4). All but 1 were associated with accidental trauma. The 1 case attributed to operative injury appears to be similar in character to cases in adults reported by Millbourn,[39] Perryman and Hoerr,[44] Brown,[9] Warren,[57] and Dunphy and co-workers.[21] It has

not, however, heretofore been considered as a postoperative complication in infants. Pseudocyst is generally considered as occurring more often in pancreatitis due to trauma than in other etiologic forms, and the occurrence of this complication in 7 of the 15 cases (46.7 per cent) shown in table 4 is consistent with such a conclusion.

As would be expected, vascular disease does not seem to play a significant role in acute pancreatitis in infancy and childhood, and no cases were found which could be attributed to a primary vascular etiology.

Thal and Brackney[53, 54] have emphasized the possible role of allergic factors as a cause of pancreatitis, particularly through the mechanism of the Arthus or Shwartzman phenomena. Because other factors appeared to be more predominant, we have listed no cases as occurring on an allergic basis. In several, however, allergy may have played some role. In the case reported by Stickler and Yonemoto[51] the patient had acute tonsillitis treated with chlortetracycline. In the course of treatment she developed urticaria, which disappeared after Benadryl therapy, but these events preceded the onset of abdominal pain. One of our patients and 1 reported by Collins[11] developed thrombocytopenia which could conceivably also have occurred on an immunogenic basis, although there is no direct evidence to support such a possibility.

TABLE 5

Idiopathic pancreatitis

Author	No. of Cases
Phélip,[45] 1920	1
Vogel,[56] 1924	1
Holzmann,[31] 1927	1
Mackenzie,[37] 1930	1
Covaro,[13] 1931–1932	1
Foged,[23] 1932	1
Desjaques,[14] 1932	1
Sujoy,[52] 1936	1
Arana and Lagos Garcia,[4] 1940	1
Anavitarte,[2] 1943	1
Dey,[15] 1951	1
Ger,[25] 1954	1
Claireaux,[10] 1956	1*
Haigh,[29] 1956	2
Blumenstock,[6] 1957	3†
Total	18

* Superimposed on fibrocystic disease of the pancreas.

† One with pseudocyst.

TABLE 6

Frequency of various etiologic groups

Etiologic Group	No. of Cases	Per Cent
Metabolic-toxic-chemical	24	32.4
Idiopathic	18	24.3
Traumatic	15	20.3
Infectious	10	13.5
Obstructive	7	9.5
Vascular	0	0
Allergic	0	0
Total	74	100.0

There were 18 cases (24.3 per cent) lacking any evidence of an associated etiology and these were placed in the idiopathic group (table 5). Although this is proportionately smaller than that shown in the Stickler and Yonemoto grouping,[51] it is, nevertheless, the second largest group even by our classification.

In a previous publication[7] we have shown that the frequency of pancreatitis on an etiologic basis, considering all age groups, follows this order: idiopathic, infectious, obstructive, vascular, metabolic-toxic-chemical, traumatic and allergic. In infants and children the order of frequency is somewhat different, as shown in table 6, *i.e.*, metabolic, idiopathic, traumatic, infectious and obstructive, with no cases on a vascular basis and probably none on an allergic basis.

Finally, it seems worth while to emphasize the importance of considering acute pancreatitis in the differential diagnosis of acute abdominal pain in infancy and childhood. All of the 6 cases in our own series were discovered at autopsy and acute pancreatitis was never considered clinically. This also appears to have been the situation in many of the cases previously reported in which the pancreatitis was discovered on exploratory laparotomy. It is pertinent, in this regard, that 10 of the 25 patients whom Stickler and Yonemoto[51] placed in the idiopathic group died of their pancreatitis, 8 of these after surgery. This would indicate that surgery may be detrimental in acute pancreatitis in infants and children, and conservative therapy preferable.

The latter can be properly directed only when the clinical diagnosis is correct, and this requires that acute pancreatitis be considered in all infants and children with acute abdominal symptoms and that the proper laboratory tests (serum amylase and lipase levels) be chosen for establishing or excluding such a diagnosis.

SUMMARY

Six cases of acute pancreatitis in infants, discovered at autopsy, have been presented. This brings to 74 the number of cases which we have been able to find in the literature. In several of the present cases the disease appeared to have been initiated in intrauterine life. All 74 cases have been grouped according to associated etiologic factors and discussed primarily from the point of view of etiologic considerations.

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EVALUATION OF PALLIDOTOMY AND THALAMOTOMY IN ABNORMAL MOTOR MOVEMENT

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Operative techniques to destroy parts of the basal ganglia and thalamus in patients with abnormal motor movements have been utilized for over 30 years. Because of the recent enthusiam and the increase in the number of thalamotomy and pallidotomy procedures, a study of the known anatomic facts on which these operations are based is deemed advisable, as well as a review of the patients selected for surgical treatment, and the results in these patients. From 1930 until 1955 operative interference was not extensively employed because of the technical difficulties involved and the relatively high operative mortality. Cooper,[2] however, introduced a method of chemopallidotomy, and following this, the procedure became more popular.

Among the first definitive procedures performed, Myers[8] utilized a transventricular operative destruction of the basal ganglia, but recently has shifted to the use of ultrasound therapy, localized by stereotaxic methods. The Philadelphia group of Spiegel and Wycis[12] has used electrocoagulation with the stereotaxic instrument in the region of the ansa lenticularis. Narabayashi and others[9] have injected procaine in oil for blocking of the medial globus pallidus, utilizing stereotaxic methods. Fenelon and Thiebaut[3] utilized leukotomes. McKinney[6] developed a method of surgical section of parts of the basal ganglia or thalamus. McCaul[5] in London and Obrador[10] in Madrid have devised special cannulas for surgical, as compared with chemical, lesions in the globus pallidus and thalamus, and Mullan[7] in Chicago has reported on the effectiveness of radioactive isotope implantations. Paxton and Dow[11] treated 53 patients for dystonias by chemopallidectomy.

When we first approached this problem we did not favor the method of Cooper and the various procedures devised for chemopallidotomy because of the inability to control the spread of the chemicals involved, and a modified McKinney method has been utilized exclusively in our series of cases. We feel the most effective lesions are those in the lateral thalamus on the border of the internal capsule (fig. 1).

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METHOD

The surgical technique used in our series consists of a vertex approach to the lateral thalamus or the basal ganglia. With the patient under local anesthesia, 50 cc. of air is first introduced into the spinal canal with withdrawal of 30 cc. of fluid. It is necessary that the pneumoencephalogram adequately delineate the 3rd ventricle and the intraventricular foramen of Monro. We are now using ventriculography instead of pneumoencephalography. The patient is then placed on the operating table and, after the usual drape, large trephine holes are made directly over the coronal suture. By direct visualization a cannula is passed downward, aiming for a point 0.5 cm. lateral to the inner canthus of the eye and 3 mm. behind the external auditory meatus, and is introduced to a point 9.7 to 10.4 cm. from the needle holder, depending on the size of the skull. X-ray films are then taken and compared with standard skull films that have been taken on the operating table before the procedure. Three measurements are then determined: (1) the position of the cannula halfway between the foramen of Monro and the pineal gland; (2) an angle of 49° to 51° from the anterior clinoid process drawn from a base line (fig. 2); and (3) the relationship of the needle to the internal auditory meatus and the coronal suture, as well as the distance of 16 mm. from the midpoint of the 3rd ventricle or the falx. The location of the needle, or cannula, is checked and rechecked until a satisfactory localization is obtained (figs. 3 and 4). Following this, cuts are made at 12:00 o'clock, at 1:30, at 3:00, at 4:30, at 6:00, at 7:30, at 9:00, at 10:30 and at 12:00 with the cannula rotated and the piano wire "blade" opened. The patient is carefully examined after each cut to determine, if possible, which actual fiber cell section is responsible for

FIG. 1. Anatomic location in horizontal section showing thalamus, capsule and globus pallidus.

the alleviation of the tremor as well as the spasticity. On three occasions cuts have been made at several depths, *e.g.*, 9.7, 9.9, 10.1 cm. (figs. 5 and 6). In 2 cases complete repositioning of the cannula posteriorly or anteriorly was carried out after initial sections were ineffectual. The operation is first done on one side, and in 3 weeks to 1 month afterward it is repeated on the contralateral side, if indicated. The procedure is always done with the patient in the supine position. The total procedure takes approximately 1 to 2 hours. We have had no complications in our series and no wound infections or other difficulties. Lethargy may be present for 48 hours.

SELECTION OF PATIENTS

In our series there have been cases of Parkinson's syndrome, unilateral and bilateral, as well as chorea, athetosis or choreoathetosis of both acquired and congenital type. Patients with Parkinson's disease showing mental symptoms or obvious senility should not be subjected to surgery. We have not operated on any completely bedfast patients. It has been determined by a number of neurosurgeons that elderly debilitated patients with arteriosclerosis usually should not be subjected to surgery. Surgical interference should be utilized when the patient has definite difficulty in working or requires help in the common everyday facets of human existence, after adequate medical treatment has been unsuccessful. We have had 1 patient in the younger age group who was almost bedridden (a 49-year-old white man) and he had a successful result following thalamotomy.

RESULTS

Review of the cases leads to the belief that three components of the Parkinson's syndrome are altered by the pallidotomies and thalamotomies. The most striking results are obtained in the plastic rigidity, and fair to excellent results have been obtained in alleviating the pill-rolling tremor, depending on where the lesion is made. The third change has been the alleviation of the depressive symptoms that accompany the Parkinson's syndrome. In 1 case this was particularly striking, and one of the observers believed this to be partially responsible for the improvement in the patient's condition. This has been of particular interest to us because of the known aggravation of the tremor by any situation that produces nervous tension.

CASE REPORTS

Case 1

A 52-year-old white woman was first seen on March 31, 1959, complaining of a tremor of the right side of the body. This had started approximately 10 years before. The patient had been seen at the Barnes Hospital and Clinic in St. Louis, and at the Mayo Clinic. She was given medication but had received no other treatment. The tremor on the right side had gradually become more severe; it involved the right arm to a greater degree than the right leg and foot, and was present even during sleep on some occasions. The patient dragged the right foot. She had no other complaints in relation to headache, anosmia, tinnitus, or other symptomatology.

Past history revealed no history of trauma to the head, back, or neck. She had no history of influenza. She had had an appendectomy and cesarean section. Her father and mother were living and well. Family history was noncontributory.

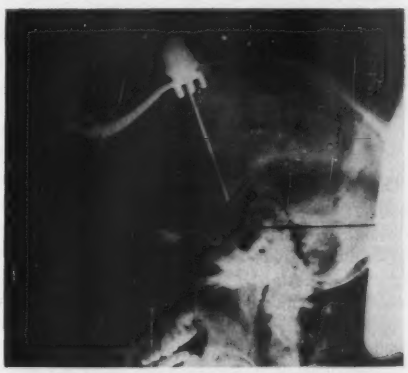
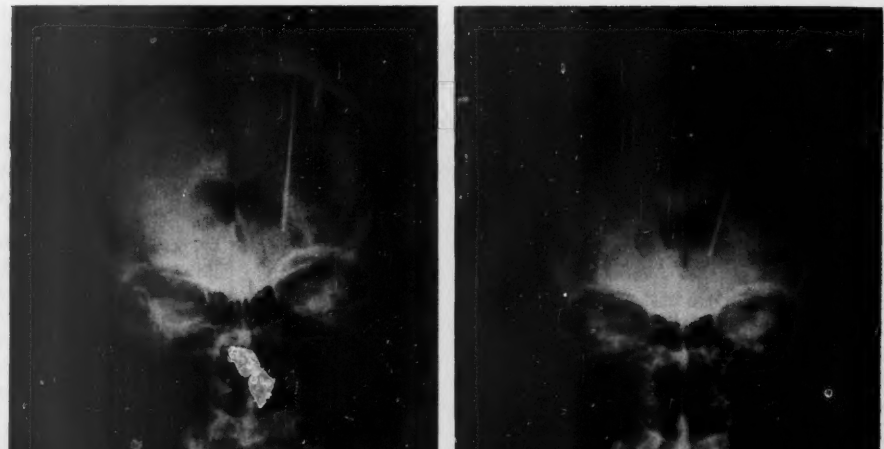


FIG. 2. X-ray showing angle from base line to produce anterior and inferior section. Horizontal short line, *B*, is most effective center point for lesion for tremor.



FIGS. 3 to 6

FIG. 3 (*top left*). Needle position is too far lateral

FIG. 4 (*top right*). Reposition of needle with center 12 to 13 cm. from midline

FIGS. 5 (*bottom left*) and 6 (*bottom right*). Needle cuts made at depths of 9.7 and 10.1 cm.

Detailed physical examination revealed a healthy, cooperative white woman. There was minimal arteriosclerosis present. Examination of the cranial nerves was negative. General neurologic examination in regard to the sensory and cerebellar pathways was normal. The patient had a marked pill-rolling, regular, rhythmic tremor which was aggravated by nervous tension and, to a certain degree, by intention movements. The tremor was present to a lesser degree in the right leg. There was moderate spasticity present in the right upper and lower extremities, with minimal cogwheel type of rigidity. The patient's gait was characterized by a dragging of the right foot without any circumduction.

The patient was admitted to Hillcrest Hospital on April 14, 1959, and a left pallidectomy was carried out first at a depth of 9.8 cm. without any definite improvement. The depth was then changed to 9.9 cm., and after this there was definite improvement in the tremor. The cuts were made at the second depth at 10:30, 12:00, and 1:30. The depth was then moved to 10 cm., and cuts were made at 12:00, 10:30 and 1:30.

The patient was discharged 5 days after the left pallidectomy. She showed marked improvement and was able to write and drink coffee with her right hand, which she had not been able to do for 4 years. She started cooking and working in the kitchen again on May 1, which she had not done for 3 years, because she had been afraid she would burn herself and break things. One of the most interesting features was the fact that her husband was very pleased that the bed did not shake at night, because the arm no longer shook.

Case 2

This 34-year-old white woman was seen on July 16, 1959, complaining of constant headache and a tremor and numbness of the right arm. The patient had had mild constant headache for years. Pneumoencephalogram was obtained in October 1958, and showed focal cortical atrophy in the left parietal lobe. The headache tended to involve the left half of the head and was of the migrainous type, associated with intermittently blurred vision, some dizziness and nausea. Occasionally, there was very severe occipital neck pain with the headache. The tremor of the right arm had been present for a period of approximately 16 years, had been originally largely of intention type, associated with some tremor at rest. It had gradually become more severe, and during the 6 months before our first examination was definitely incapacitating her in her work as a beauty operator. The numbness of the right arm had been noted for a period of 2 to 3 years, and had involved all modalities of sensation.

Past history revealed that the patient had had an injury at school when she was hit on the head by a baseball. She was unconscious intermittently for a period of 2 days, but had no specific treatment. She recovered from this injury apparently without residuals. The present illness started approximately 8 years after the accident. Past history also revealed that the patient had had her tubes ligated. She had no serious illnesses or operations. She had had the usual childhood diseases.

Detailed examination showed a healthy, cooperative white woman in no distress. There was a marked intention tremor and a pill-rolling type tremor with the hand at rest. There was numbness of all cortical modalities of sensation over the right upper extremity, with very minimal loss of pain and temperature but a rather marked loss of number recognition, two-point sensation, and ability to distinguish various types of cloth. The patient had no papilledema. The reflexes were slightly increased over the whole right side of the body as compared with the left. Blood pressure was 108/70.

Detailed electroencephalographic studies revealed a slight abnormality, manifest by sharp spike wave formation and slow wave activity of minimal degree in the left posterior parietal region superiorly.

It should be noted that intake of alcohol would often stop the tremor, and that the tremor would be worse when the patient was very tired and under tension.

The patient was admitted to Hillcrest Hospital on August 2, 1959, and a series of stellate ganglion blocks was performed on the left side. After each block the patient had definite relief of her tremor and numbness for a period varying from 25 minutes to 3 hours. Partial relief would be obtained over a period of approximately 24 hours. Consequently, on August 10, 1959, a bilateral superior cervical sympathectomy was performed, and the patient was discharged 4 days later. Immediately after surgery, the tremor was almost completely alleviated, and this has persisted, for a period of 9 months. The patient's numbness entirely disappeared and has shown no evidence of recurrence. There has been no change in the improvement in the tremor from the immediate postoperative period up to the present time. Electroencephalographic studies showed definite improvement. The patient is troubled to a certain extent with increased stuffiness of the nose. She does not mind the ptosis.

Case 3

R. W., a 70-year-old white man, was admitted to our department on April 19, 1960, complaining of severe tremor of the right and left arm and hand, and of both legs. He had had marked difficulty in talking and increasing difficulty in getting about. He had difficulty in getting out of bed, and during the last 2 years had been unable to button his clothes. His legs had become "very stiff" during the last year, and it had been difficult to ambulate.

Detailed neurologic examination showed typical Parkinsonian syndrome with cogwheel rigidity and marked tremor, made worse by excitement or exertion. There was swelling and redness of the midportion of the upper left eyelid and increased injection of the conjunctivas of both eyes. The heart, lungs and abdomen were essentially within normal limits. There were no signs of increased intracranial pressure. There was a very slight pitting edema of the dorsum of both feet.

A left thalamotomy was performed at a depth of 10.4 cm., and with the cutting instrument set at 10:30 (fig. 7), complete relief of the tremor of

FIG. 7. Needle in position for medical thalamotomy.

the right hand immediately supervened. After this several lateral cuts were made, and the patient was returned to the ward in good condition. He was discharged completely relieved of tremor 4 days after his surgery. The patient was to be readmitted to the hospital on June 9, 1960, for a right thalamotomy.

DISCUSSION

Many questions have been raised by members of the department after reviewing the literature on this subject and in studying the results in a large number of our cases of tremor, treated medically and surgically by various means in the past. One wonders what physiologic change is produced by the chemopallidotomies and surgical procedures utilized in the treatment of Parkinson's syndrome, dystonia musculorum deformans, athetosis and chorea. In 1924 an Australian surgeon, N. D. Royle, showed that superior cervical sympathectomy would alter spasticity in the human. A series of cases was studied and treated by Gardner and Karnosh[4] and Stowell[13] at the Cleveland Clinic. It was noted that for a period of 1 to 2 days after superior cervical sympathectomy there was complete alleviation of tremor in Parkinson's syndrome, and that in a very few cases there was permanent improvement in spasticity. One of the cases of unilateral tremor, associated with cortical atrophy in the parietal lobe, was permanently altered by superior cervical sympathectomy.

Anatomic studies by Brierley and Beck[1] in England have shown that any study of the spatial relationships in the brain is difficult to evaluate, as there are widespread individual variations in the anterior, posterior and vertical position of the posterior commisure, and that the posterior commissure was found to bear no constant relationship to the thalamic nuclei in the anterior, posterior, or vertical plane. The anterior-posterior range of the position of the posterior commissure was 26 to 41 per cent of the total thalamic length. This difference represented a 5.3-mm. thalamic length in an average total thalamic length of 34.5 mm. Considerable variation was demonstrated in the lateral and vertical coordinates of the globus pallidus. The paramount importance of individual variations was stressed in regard to limitations of accuracy in human stereotaxic brain surgery. White and co-workers[14] at the Mayo Clinic discussed a case of bilateral pallidal destruction resulting from a unilateral chemopallidotomy with bilateral destruction of the medial portion of the globus pallidus as determined by autopsy findings. This particular patient had no favorable alteration or improvement following this procedure. Rigidity and tremor were unchanged. The authors concluded that "there is a decided lack of a precise understanding of the physiological significance of pallidal function in paralysis agitans."

This brings up the question of exactly what is being done by sectioning the fibers or destroying cells in the globus pallidus in the thalamus. It is becoming evident that operations in the region of the thalamus are more effective than those in the region of the globus pallidus. This was strikingly demonstrated in Mullan's report[7] on 24 patients treated by alcohol injection or radioactive isotope necrosis. He showed that an area in the lateral thalamus near the junction of the internal capsule, approximately 2.5 cm. behind the anterior commissure, is the most effective location for altering tremor. It was interesting to note that in 2 of his cases there was a very marked alleviation of pain. The author definitely preferred isotope implantation to alcohol injections. He concluded that lesions produced in the globus pallidus were disappointing. If one analyzes, however, the reported cases and the apparent location of the lesions, one concludes

that a number of different locations or surgical lesions may produce favorable or unfavorable results, both in regard to the tremor and to the spasticity.

It is concluded that a number of cellular areas and tracts enter into the production and alteration of the tremor and spasticity of Parkinson's syndrome and that sensory (afferent) impulses may be of great importance. I feel that operative procedures have been successful and should be continued. I favor the leukotome, or piano wire type of section, rather than the chemical procedure because of the controllability of the cuts as compared to the diffusion of a chemical in the brain. We feel that as many anatomic studies as possible should be carried out, with very carefully detailed movies of these patients, as well as an evaluation of the eventual pathologic findings by trained observers. Last, more studies should be conducted of other parts of the central nervous system and their relationship to this particular tremor. The physiologic and anatomic connection between the basal ganglia, thalamus and cerebellum are well known, as is the fact that many components of the cerebellar tremor are similar to the so-called basal ganglia tremor. One should also consider further studies into the influences of the superior cervical sympathetic chain on tremor and alteration of muscle spasticity.

SUMMARY

A small series of patients was subjected to unilateral and bilateral operative procedures for attempted relief of tremors, spasticity and choreoathetosis. Multiple sections were made at a one- or two-stage procedure, with good results in the thalamotomy group of cases and poor results in the pallidotomy group. Thalamotomy is of definite benefit in altering the spasticity and tremor of Parkinson's syndrome, whereas pallidotomy is apparently effective only in severe muscle spasticity. Both procedures help acquired athetosis and choreiform movements. Further studies are necessary for a pathophysiologic explanation of the failures and the "cures."

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SELECTIVE CINEANGIOCARDIOGRAPHY*

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Until the advent of image intensification cineangiocardiography was not satisfactory because of poor quality reproduction and high x-ray dosage to the patient. The development of fast black and white movie film with fine grain quality has aided the production of excellent cineangiocardiography at very fast shutter speeds and low x-ray exposure of the patient.

In our laboratory a Picker 500, 5-in. image amplifier x-ray machine is used with cine taken at 60 frames per sec., using Kodak green sensitive (Cine Flure) type film. The volume of dye injected varies from 5 cc. to a maximum of 2 cc. per kg. of body weight, depending upon the lesion to be shown. Hypaque 50 per cent is used as contrast medium. Using 120 kv. and automatic brightness control, the usual exposure of 25 ft. of film at 60 frames per sec. delivers 7 r of surface dose to the patient.[1]

After the usual history, physical examination, electrocardiogram, cardiac fluoroscopy and catheterization with oximetry and pressures, one is usually able to arrive at an anatomic and physiologic diagnosis. Cineangiocardiography has been found very helpful for further elucidation of anatomic deformity when surgery is considered. Occasionally cineangiocardiography is used as the definitive diagnostic procedure in severe cyanotic heart disease in infancy to aid in the evaluation of the origin and size of pulmonary arteries, as, for example, before palliative Blalock shunt operation. Usually, however, this method is used as a supplemental diagnostic procedure to confirm and further evaluate anatomic congenital defects.[2] Consideration of some of the different congenital deformities will illustrate our use of this diagnostic procedure.

CYANOTIC HEART DISEASE

Dye dilution studies, pressure curves and oximetry frequently give an inconclusive diagnosis. Cineangiocardiography can be used very effectively to differentiate cyanotic anomalies by injection of dye into the right ventricle with observation of the ventricular septum and outflow tracts.

In transposition the dye immediately fills the aorta coming from the right ventricle. With tricuspid atresia the small or absent right ventricle is obvious and the level of the right to left shunt is seen. Single ventricle is evident from absence of the ventricular septum. Truncus arteriosus and its many different forms can usually be evaluated.

Tetralogy of Fallot is of much more importance surgically since complete repairs have been effected. The type of pulmonic stenosis and size of pulmonary artery is of prime concern to the surgeon who wishes to attempt complete repair. Pulmonary artery size is important when considering palliative surgery such as Blalock-Taussig anastomosis. An atretic pulmonary artery beyond the bifurcation almost precludes any type of repair. Cineangiocardiography is the only method which can effectively warn the surgeon preoperatively that a noncorrectable situation is present and it will aid in the preoperative planning of corrective surgery on the right ventricle out-flow tract in correctable cases.

ACYANOTIC HEART DISEASE

The diagnosis of atrial septal defects is seldom a problem. The size and location of ostium primum and secundum defects can be well demonstrated by injecting dye into either left or right auricle with the patient in the 30 per cent left occipitoanterior position. The position of anomalous pulmonary veins can also frequently be determined and their relationship to the auricular septum demonstrated preoperatively.

Persistent common atrioventricular canal defects can usually be well demonstrated by dye injection into the right ventricle. The reflux of dye back into the right and left auricles and the demonstration of the defect in the ventricular septum makes the diagnosis complete.

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Isolated ventricular septal defect can be demonstrated with cineangiocardiography at camera speeds of 60 frames per sec. even though the physiologic shunt of blood is entirely left to right. We have never failed to demonstrate the size and location of the ventricular septal defect, as increased pressure in the right ventricle during injection will force dye across the defect in ventricular diastole.

Those cases that have both pulmonary stenosis and ventricular septal defect present a difficult diagnostic problem when the right ventricle and left ventricle pressures are nearly equal so that oximetry and dye curves fail to reveal the septal defect. In all cases of pulmonary stenosis, cineangiocardiography is done to demonstrate the type of pulmonary stenosis, valvular or infundibular, and to exclude associated ventricular septal defects. With pure pulmonic stenosis the surgical approach through the pulmonary artery, infundibular chamber or lower ventricle can best be preoperatively determined by angiography. Combined infundibular and valvular pulmonic stenosis is most difficult to diagnose by the usual pressure curves, as the infundibular stenosis will frequently effectively damp out the change in pressure at the valve orifice even if the catheterizing surgeon is fortunate enough to get the catheter through both. Angiography may help considerably in this diagnostic problem and is of prime help when it is not possible to pass the catheter through the right ventricle out-flow tract.

The diagnosis of patent ductus arteriosus usually need not be supported by cardiac catheterization except to rule out associated defects and to preoperatively measure pulmonary artery pressure. The differentiation between a patent ductus and pulmonary-aortic window can effectively be done with cineangiocardiography. If the catheter enters the aorta but the site of entry is in question, a small dye injection will reflux back through the communication, and its site determined. If the catheter does not pass through the defect the injection is made in the right ventricle and de-opacification of the pulmonary artery with blood from the aorta will demonstrate the level of communication.

Coarctation of the aorta has been well demonstrated in this laboratory by cineangiography with injection of dye into the right ventricle and observation of the aortic arch.

Pulmonary arteriovenous fistula can be well demonstrated as to location and extent by cineangiography. The field of examination can be studied with injection only in the right ventricle.

Other cardiovascular disease processes such as coronary disease, aortic and carotid aneurysms, renal artery and aortic occlusive disease can well be demonstrated by right heart injection and selective cineangiography of the part involved. Left atrial tumors have been very well demonstrated in other laboratories. Congenital mitral stenosis has been demonstrated in our laboratory with right heart injection of dye.

SUMMARY

1. Cineangiocardiography is an integral part of any cardiovascular diagnostic laboratory.

2. Selective cineangiocardiography for preoperative surgical evaluation of congenital heart disease has been discussed.

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THE SUPERIOR AND INFERIOR LARYNGEAL NERVE AS RELATED TO THYROID SURGERY

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The technical problem in thyroid surgery is an anatomic one. This relates to the patient's ability to be comfortable, to speak, and to carry on his profession of speaker, orator, singer or common citizen without embarrassment or encumbrance. A full recognition of the anatomic relation of the superior and inferior laryngeal nerves to the structures encountered in thyroid surgery is of the utmost importance in obtaining postoperatively an unimpaired vocal expression. It is for this reason that we examine preoperatively and postoperatively the vocal cords of every thyroid patient upon whom we operate. It is also a good medicolegal protection.

We have constantly kept in mind the physiologic function of the superior laryngeal nerve, and have tried consistently to avoid injury to the main branch or to either of its two divisions. We have recognized the inadvisability of trying to expose it at the time of operation, because of technical difficulties, in contrast to exposure of the recurrent laryngeal nerve, which if cautiously and skillfully done adds much to the preservation of its function.

We have made it a policy to ligate only the superior thyroid artery and vein and have refrained from encompassing wide sweeps of adjacent tissue in superior thyroid pole ligation. This might well include the nerve in its entirety or one or the other of its two branches, the internal or external laryngeal nerves. In spite of these precautions as related to the superior and inferior laryngeal nerves, we are not without error. Through these precautions, nerve injury has been reduced to a minimum and is far less than before we became conscious of the importance of exposure of the recurrent laryngeal nerve at the time of operation; and by our cautious ligation of the superior thyroid artery and vein and blunt dissection of the superior thyroid tissue we have tried to protect the superior laryngeal nerve and either of its divisions.

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Although I had repeatedly, at autopsy, dissected out both of these nerves and assured myself of their anatomic relationship to other structures that pertained to thyroidectomy, I had not fully appreciated the importance of the superior laryngeal nerve as it pertains to voice volume until I heard and read the very excellent article, "The Superior Laryngeal in Relation to Thyroid Surgery," by Leon F. Hirzel[2] of Bay Amo, Cuba, presented at the 1957 Pan-Pacific Surgical Association at Honolulu, Hawaii. I advise all who are interested to read it. It presented many phases of post-thyroid voice volume and tone that I had repeatedly spoken about and urged; but it brought out many facts which I had not observed or thought to be pertinent to superior laryngeal nerve injury, for example, strangulation on deglutition and cough.

THE SUPERIOR LARYNGEAL NERVE

Rarely is damage to the superior laryngeal nerve mentioned in any discussion of thyroid surgery, but much emphasis is placed upon the preservation of the recurrent laryngeal nerve. This, the superior laryngeal nerve, lies so close to the superior thyroid vessels that either it, or one of the two divisions, the external or the internal, may be damaged in the process of ligating and dividing the superior thyroid artery and vein. These nerves may be traumatized or injured unless there is precaution concerning their preservation in dissection of the superior thyroid vascular structures.

This anatomy must be kept in mind. The superior laryngeal nerve starts at the ganglion nodosum of the vagus nerve and passes downward very close to the superior thyroid artery, where it divides into the external and the internal superior laryngeal branches.

The external branch passes closer to the superior thyroid artery than the internal branch and is therefore more vulnerable. It is a nerve of very small caliber and supplies the constrictor and cricothyroid muscles. The muscles have the function of providing tension or tone to the vocal

cords during phonation. They are factors that produce volume and clarity of speech. Often after thyroidectomy, volume and pitch of voice are lost, and this is not understood because the cords are seen to move on laryngoscopic examination. We have seen cases of external laryngeal nerve damage with a shallow tiring voice, without volume, and the cords on laryngoscopic examination showing loss of tone. They moved properly, but were wavy or flabby. In our observation most of these improve either from restoration of nerve function due to nerve regeneration or to a reasonable degree of laryngeal compensation.

The internal laryngeal nerve enters the larynx through the thyrohyoid membrane and supplies innervation to the larynx and epiglottis. There is a descending branch which joins the ascending branch of the recurrent laryngeal nerve. Trauma to the internal superior laryngeal branch will result in a severe persistent cough after deglutition but with no alternation of phonation (fig. 1).

Fewer patients have evidence of damage to the internal laryngeal because its position is more distant to the superior thyroid vessels and is less frequently damaged in superior pole ligation. Paroxysm of coughing after ingestion of liquids results from internal laryngeal nerve involvement. In some instances this is diminished or abolished by placing the head in certain positions.

THE RECURRENT LARYNGEAL NERVE

The recurrent laryngeal nerve with few exceptions presents itself in the neck, coming up out of the chest and ascending to the horn of the thyroid cartilage and disappearing into the larynx below the inferior constrictor muscle. It innervates the posterior arytenoid and the lateral arytenoid muscles, which control abduction and adduction of the vocal cords. The nerve as it ascends in the neck is in relation to the inferior thyroid artery. The nerve usually passes upward to the larynx mesial or beneath the inferior thyroid artery, but may be lateral or pass through the dividing tributaries of the artery. If lateral, traction on the gland puts it on tension and it can be felt as a tense, string-like cord. In this position it is easily exposed. If the nerve is under the artery, elevation of the gland does not disturb the anatomic position. Because of its relation to this artery it is important to first locate the trunk of the artery as it emerges from behind the common carotid artery before attempting to visualize the nerve. It is wise to visualize the recurrent laryngeal nerve before ligating the inferior thyroid artery. The nerve is best approached through the areolar tissue below the inferior thyroid artery between the common carotid artery and the trachea and esophagus. It is essential that this be done under a bright light and it must be in a dry field free from bleeding. In finding the nerve, the exploring instrument is gently opened, always in the direction of the course of the nerve. It should be done very gently and the nerve should never be stretched or traumatized. Usually, by careful exploration, there will be little or no bleeding and no technical difficulty in exposing the recurrent laryngeal nerve. The nerve is recognized as a pearly looking strand of longitudinal nerve fibers running upward along the tracheoesophageal groove in the direction of the larynx. The nerve is first exposed in the lower part of the neck and gently traced to the point where it enters the larynx. At this point it is most likely to be enclosed in a suture or ligature. Extreme care should be taken to avoid injury or trauma. Often it is seen dividing into two sections, the smaller section representing the abductor fibers and the larger section representing the adductor fibers. Many divisions have been mentioned. The nerve is rarely injured in the lower part of the neck, but usually at the point of entrance into

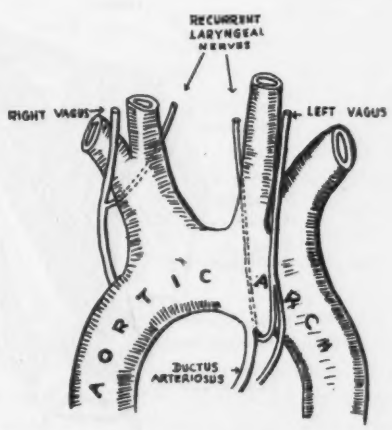

FIG. 1. Relations of vagus and recurrent laryngeal nerves to the great vessels. Notice the oblique position of the recurrent nerve on the right as contrasted to the direct position on the left. This is of surgical importance.

the larynx, and frequently when attempts are made to stop a bleeding point by hemostat, ligature or by suture. It is necessary, therefore, to expose the nerve in its entire course in the neck; this can be done without trauma or nerve injury if caution is exercised. It is well to remember that the nerve on the left side ascends in a parallel relationship to the trachea and the esophagus, whereas on the right side, because of its recurrence around the first part of the subclavian artery or innominate artery, it is in a slightly diagonal position in the lower part of the neck as it emerges from behind the common carotid artery (figs. 2 and 3).

It is just as essential, in thyroid surgery, to expose the nerve as it is in gall bladder surgery to expose and visualize the extrahepatic bile ducts and to isolate the cystic artery and see its relation to the right hepatic artery before ligation and division of either the cystic duct or cystic artery. In my opinion, the two procedures are comparable. The ureters in certain pelvic conditions are comparable.

The nerve is never preoperatively paralyzed because of thyroid pathology except in some instances of extensive thyroid malignancy or deeply placed intrathoracic goiter. Even in large cervical goiters or intrathoracic goiters, the nerve, as I have observed it, is not involved. Visualization is an avenue of protection and the only procedure to determine a normal or abnormal anatomic position. If abnormal, it can be determined only by exposure and it is highly essential to know this before proceeding with the operation. If the recurrent nerve is not exposed at operation, it can only be hoped it has not been injured.

Postoperative suture of a severed recurrent laryngeal nerve is not satisfactory. Gregg[1] states: "They can be ligated, provided the suture is removed within a few hours. . . return of function will be observed in a matter of weeks."

There is no instance in which we were unable to expose the recurrent laryngeal nerve. We found no considerable variation in its relation to the inferior thyroid artery or its branches, and no invasion of the nerve into the substance of the thyroid gland.

I have seen but few of the numerous anomalies that have been frequently mentioned and I have never seen an inferior laryngeal nerve come off directly from the vagus to the thyroid gland. It has been recorded. There was one instance of incomplete descent of the recurrent nerve on one side which recurred around the inferior thyroid artery.

We have had no permanent bilateral cord paralysis. There was 1 patient, because of edema

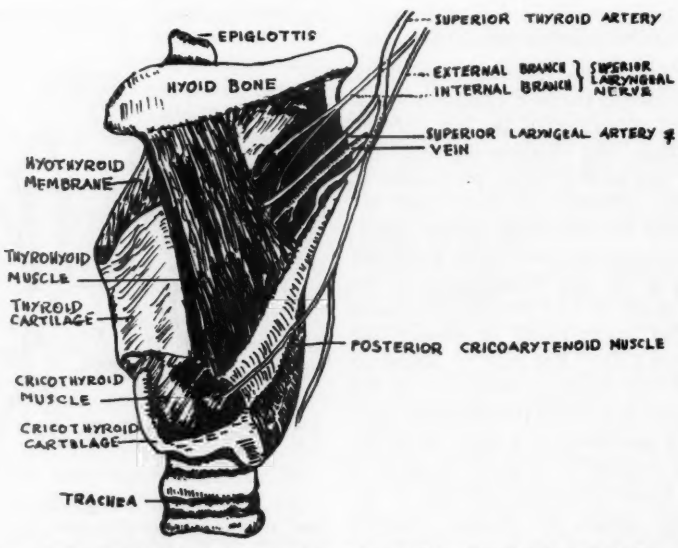

FIG. 2. Larynx, showing the intrinsic laryngeal muscles and the distribution of the laryngeal nerve

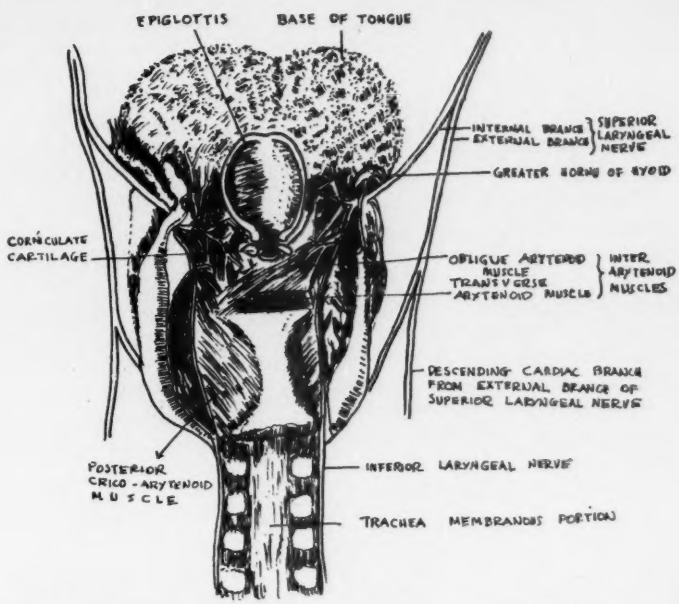

FIG. 3. Dorsal view of larynx, showing the intrinsic laryngeal muscles and the distribution of the laryngeal nerves.

and severe tracheitis, who showed, on laryngoscopy, immobile edematous cords that required tracheostomy. Complete recovery resulted and the cords ultimately moved normally.

Not all patients with unilateral cord lesions are hoarse. Most will show only a temporary paralysis which may last 6 weeks to 3 months. Most paralysis, if careful surgery has been done, is the result of edema, trauma and hematoma. If one is careful with nerve exposure, suture and ligation, there will be few irreversible nerve injuries.

Hoarseness and nerve injury are not synonymous. Hoarseness without nerve injury is common, and nerve injury without hoarseness may occur, but it is rare.

Compensation by the uninjured nerve usually temporarily overcomes any change in voice. The cord fibrosis, associated with cord fixation, will eventually result in breathing difficulty and voice deficiency. The airway is lessened and a stridor may be experienced on exertion. The voice often becomes cracked or off tone on vocal exertion.

Bilateral nerve injury is more serious from the viewpoint of breathing and limitation of physical effort. The lost voice will partially return in a few months; but the cords will progressively undergo atrophy fibrosis, and as they approach each other in the midline, there will be a resulting narrowing of the glottic space and consequent shortness of breath on even mild exertion.

It is difficult to estimate the injuries relating to the superior laryngeal nerve. Patients have sometimes complained of cough and respiratory effort after taking fluids. In my observation these clear up in a relatively short time. At least they become less annoying. Some patients have deficient tone and impaired volume which persists in the presence of laryngoscopic evidence of vocal cord function. This can only indicate impairment to the external branch of the superior laryngeal nerve.

In 880 cases, in which we have observed the vocal cords by laryngoscopic examination after operation, we have found 21 instances of unilateral cord paralysis. Of these 17 proved to be transitory, and returned to normal after a period of 3 to 4 months. These obviously were not due to nerve injury but to edema from trauma of surgery. Four cases were permanently paralyzed

and developed progressive voice changes and a slight respiratory stridor.

SUMMARY

We advocate careful preservation of the superior laryngeal nerve by ligating only the superior thyroid vessels at the time of surgery and by the avoidance of encompassing wide scopes of surrounding tissue.

We advocate the exposure of the recurrent laryngeal nerve in all thyroid operations except lesions of the isthmus or discrete adenomas in the anterior portion of the lateral lobes.

The effects of damage to both the superior and the inferior laryngeal nerves have been discussed. Methods of protection have been mentioned.

Acknowledgements. I acknowledge with thanks the opinions expressed, in correspondence, by Drs. Joel Baker, George Crile, Jr., Bentley Colcock, Ralph L. Gregg, Arnold Jackson, John McClintock, and Robertson Ward, who so kindly replied to my interrogations. I am glad to state that their approach to this problem is, in large measure, similar to mine.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS

A Review of the Literature and Report of Two Cases

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Spontaneous rupture of the esophagus is a little known entity and is not even mentioned in many of the modern surgical texts. This, however, belies its importance, for it undoubtedly occurs more often than suspected and probably is assigned a diagnosis of some acute cardiovascular accident when postmortem examination is not performed. It is undoubtedly even missed at autopsy, well hidden by empyema or other secondary changes.

It is probable that many physicians are unaware that rupture of the esophagus is one of the important surgical emergencies; attention should be focused on the problem since a missed diagnosis is catastrophic. To lose a patient with this disease is usually an error of omission and not an error of commission.

The literature goes back to Boerhave's classic description of the Baron Von Wassenaer, the grand admiral of Holland. The Baron was indulging in the old Roman custom of engorging himself to the point of discomfort, and then inducing regurgitation so that he could return to enjoy more gluttony. Boerhave found the distal esophageal tear at postmortem examination. Moynihan[5] has traced 200 cases in the literature to 1954, most of them having been reported in the last two decades.

PATHOGENESIS

Choice of the term "spontaneous" has been cited by several writers as a misnomer. The set of circumstances that occur in most of these cases is similar and suggests a common etiology, not in pathology but rather in initiating physicodynamics.

The great majority of cases are related to vomiting, and the term "postemetic" rupture has been suggested as more descriptive. Overindulgence in both food and alcohol is the usual story and a full stomach is probably a prerequisite. Some fairly violent hydrostatic forces must then be brought into play, and although vomiting is the usual causal act, cases have also been reported during childbirth, weight lifting, grand mal seizures, and defecation. The common denominator is increased intra-abdominal pressure and, in vomiting, a forceful descent of the diaphragm. Whether a food bolus becomes lodged in the esophagus or there is lack of muscular coordination, the expulsion of gastric contents into the esophagus causes a rapid and extreme pressure increase, with rupture usually occurring in the left posterolateral area of the distal normal esophagus. The tear is always described as longitudinal, and biopsy on several occasions has shown no underlying pathology of the esophagus.

Several investigators, working with fresh cadavers and with both removed and *in situ* organs, have reproduced this same picture with remarkable regularity and similarity, again in subjects with no intrinsic esophageal disease.

The only writers who dispute the initial role of physical forces in the pathogenesis are Brackney and co-workers,[2] who feel that the prime factor is an established acid-peptic erosion injury to the esophagus. Their work, which was supported by some animal experiments, is contrary to most other thinking in the literature on this disease.

CLINICAL PICTURE

The rupture of the esophagus that follows the pressure forces we have discussed produces a much more violent and dramatic clinical picture than does the perforation of a diseased esophagus during the progression of inflammatory or malignant disease. The tear is from 2 to 8 or 10 cm. in length and exposes the mediastinum to the massive insult of a full stomach which is rapidly evacuating itself. The initial pressure or the necrotizing virulent chemical and bacterial reaction usually breaks through the mediastinal pleura to evacuate into the left chest cavity, or sometimes into the right, or both.

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The initial physical findings may suggest an acute abdominal catastrophe, and there are several references to laparotomies performed for the disease. More commonly, one is attracted more to the chest symptoms and here differentiation from cardiac and great vessel accidents is necessary. When subcutaneous emphysema appears in the neck and signs of pleural fluid are present, the explanation for the severe pain, dyspnea, cyanosis, and shock becomes apparent. If mediastinal emphysema is not present and the picture is not clear, other diagnostic methods can be of help. Thoracentesis will reveal gastric fluid of acid nature or will recover blue fluid when methylene blue is given by mouth. A Lipiodol swallow will demonstrate extravasation. The patient, however, may be too sick for much manipulation and this should not be pursued if the other signs are apparent.

TREATMENT

Historically, the first thoracotomy for this disease was performed by Collis in 1943, but the patient died. The first successful thoracotomy was done in 1946 by Barrett.[1]

Earlier surgical thought was that these patients were too sick for immediate surgery and required a period of resuscitation and supportive care. There is no doubt that this reasoning is illogical in the early case. Surgical drainage and repair are the most important facets of resuscitation and this is clearly brought into focus when one sees the often dramatic improvement the moment the mediastinum is widely decompressed.

Rapid preparation of the patient will include analgesia, oxygen, massive antibiotics, and treatment of hypotension with both crystalloids and blood or adjunctive vasopressors. A large pneumothorax or effusion can be temporarily treated by closed thoracotomy prior to open thoracotomy, and a right-sided hydropneumothorax should be intubated prior to left thoracotomy.

Following these measures the left lower chest is entered and the mediastinum widely opened, if necessary, well above the aortic arch. All food particles are removed and the necrotic mediastinal pleural and areolar tissue is debrided. The esophageal tear is repaired and the chest is widely drained, utilizing controlled suction.

When the case is recognized 36 to 48 hours after rupture and there is an established empyema and some degree of stability of the situation, it may be best only to provide chest drainage and do a feeding jejunostomy. Esophageal repair this late in the course of the disease is very tenuous, with extremely friable tissue for suture.

The results of treatment have been well summarized by Samson[7] and depend on the stage of the disease at which treatment was instituted, and whether open thoracotomy and repair or only drainage procedures were done. It is clear that the best results are in those treated by repair early in their disease and that drainage procedures alone in this early stage have no place. It is also clear that those seen late or/and treated by drainage alone, if they survive will have a protracted septic course requiring multiple operative procedures. Thus, the result of treatment in those who survive is usually either rapid convalescence or the sequelae of chronic empyema, esophageal fistula, and all this implies. The over-all mortality of the problem is shown by a 1952 summary of 134 collected cases with only 27 survivals.[4]

CASE REPORTS

Case 1. A 51-year-old white man was admitted on August 15, 1957, 2 hours after the onset of his symptoms. Several hours after dinner he had become nauseated and vomited and retched repeatedly. It was immediately after one of these retching episodes that he experienced sudden severe lower chest pain radiating into the midepigastrium and lower dorsal region. Tachypnea, cyanosis, and hypotension rapidly followed and a tentative diagnosis of myocardial infarction was made. Vasopressors were necessary through the next period until a chest film was taken some 12 or 13 hours after admission. Bilateral hydropneumothorax was present, but no mediastinal emphysema. Immediate bilateral thoracentesis produced dark, highly acid fluid and the diagnosis was clear.

Seventeen hours after the onset of symptoms, and after a period of about 10 hours of hypotension despite all supportive measures, he was taken to the operating room. A tube closed thoracotomy was performed on the right side, draining 1000 cc. of gastric fluid. An open left formal thoracotomy was then done, evacuating 1500 cc. of gastric fluid . . . and food particles. Underlying a mediastinal pleural defect a 3-cm. tear in the posterolateral wall of the esophagus just above the diaphragm was found and repaired over a Levin tube. The mediastinum was widely

opened. No gross esophageal intrinsic pathology was noted, although no biopsy was taken.

After these procedures the vital signs returned to normal, but the problem from then until his death 3 weeks later was one of unrelenting lower nephron nephrosis with isotonuria, uremia, and metabolic acidosis.

The left lung promptly re-expanded. The esophageal repair broke down and a fistula drained *via* the right chest. A right pyocyaneus empyema developed, but was well drained. A feeding jejunostomy was done.

Twelve days after the initial operation massive bleeding filled the right chest and an emergency open thoracotomy on the right was performed with evacuation of a fresh hemothorax. No active bleeding was seen, but it was thought probable that eroded intercostal vessels adjacent to tube drainage may have been the source. The right lung was rapidly decorticated and a few sutures were again placed in the esophageal defect, but held very poorly. Film subsequent to this showed full bilateral expansion, and fistulous drainage became negligible. Despite all other measures he died in uremia 3 weeks after admission, the blood urea nitrogen never falling below 150 subsequent to his initial hypotension and operation. Autopsy was refused.

Case 2. A 43-year-old man vomited after eating Thanksgiving dinner in 1958: this led to excruciating abdominal and lower chest pains. A laparotomy for perforated viscus was performed locally, revealing no abdominal disease. Eleven days later he was transferred with left empyema, bilateral pneumonia, and mediastinal widening suggesting abscess—all of *Staphylococcus aureus* bacteriology. He was profoundly septic.

Following transfusions a left open thoracotomy was performed, decorticating a fibrin peel from the left lung, widely opening and debriding the mediastinum and removing turkey and vegetable material. A tenuous and rapid repair of a 4-cm. esophageal laceration was done with little hope of holding at this late date. A feeding jejunostomy was done immediately and a tracheostomy was performed the next morning.

His course showed progressive improvement with clearing of pneumonic and pleural disease and a small, well drained esophageal fistula.

Two weeks later, when recovery seemed assured, he became suddenly disoriented and showed rapid electrolyte unbalance and uremia. He died 10 days after this unexplained episode.

Autopsy showed typical gross sulfa crystals in the major renal calyces and microscopic tubular sulfa obstruction which was the cause of his demise. Sulfa was a necessary adjunct in his antibacterial regimen. The esophagus was almost healed. No pleural problems were present.

SUMMARY

The salient points in diagnosis and treatment, with a review of the literature, are presented, and 2 cases of spontaneous esophageal rupture, demonstrating many of the possible complications, are described.

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RUPTURE OF THE JEJUNUM DUE TO NONPENETRATING TRAUMA AS AN ISOLATED INJURY

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Multiple injuries in the abdomen due to nonpenetrating trauma are very common. Small bowel rupture is not rare in comparison to other abdominal injuries. Isolated injury to the small bowel without any other trauma is more rare. In the small bowel, the upper jejunum and lower ileum are involved equally as often, whereas duodenal rupture is much more uncommon. It has been speculated that the areas involved have a shorter, firmer ligamentous attachment and that they have the spinal column or the crest of the ileum as fixed points for crushing of the bowel from the external blow. Isolated jejunal rupture has been occasionally reported and usually is diagnosed at laparotomy or autopsy.

The pathology of jejunal injury with a small rent occurring in the antimesenteric border within the first 2 feet of the jejunum has been well described. Tears on the mesenteric side of the bowel and complete transverse separations have been documented together with tears of the mesentery. The blow need not be violent. Aristotle said, "A slight blow will cause rupture of the intestine without injury to the skin." It has been known that there is an "illusive calm" with lack of early symptoms.[11] This has been said to be due to lack of peritoneal soiling. There must have been reluctance in the earlier years to perform a laparotomy. Doubtlessly related to this factor are the amazing reports of the high rate of mortality from this injury as shown in table 1.[7]

No large series of mortality figures of isolated jejunal injury in the last decade is available although there are isolated reports of complete cures.[13, 15] Mortality figures show all cases, including the nonoperative ones. The operative mortality was nearly as high until the past few years. Martin and others[9] show a 2.8 per cent mortality rate for any one isolated abdominal injury in 106 patients for 10 years, ending in 1958.

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The reports on the value of x-ray are confusing. Free subdiaphragmatic air has been reported in less than 50 per cent of the cases and no other consistent findings have been noted. The presence of air under the transverse mesocolon has been described.[3, 8] Massie,[10] in 1924, was the first to note this localized pneumoperitoneum. The greatest value of x-ray is probably in ruling out other evidences of trauma, such as a retroperitoneal duodenal rupture.

In a ruptured jejunum the lack of early findings is significant. In adults the most common causes now are automobile accidents and industrial injuries;[12] in children, it is usually a blow or fall while at play.

CASE REPORTS

Five patients with upper jejunal rupture were admitted to our hospital during a 3-year interval, from October 1956 through December 1959. Three of them were operated on by one of us (W. L. C.).

Case 1. T. B., a 23-year-old steel worker, was injured at 8:30 p.m. on October 26, 1956. His left midabdomen was crushed between the ends of two large, 2-ton pipes which were being brought together. He was admitted to the hospital 4 hours after injury. Routine laboratory work was normal. On close observation he developed signs of peritonitis over a 12-hour period. X-rays then showed no free air in the peritoneal cavity.

Eighteen hours after injury a laparotomy was performed through a left paramedian incision. Approximately 10 cm. from the ligament of Treitz, on the antimesenteric border of the jejunum, was a transverse 2-cm. rent; there was also a rent in the adjacent mesentery, near it's base, 4 cm. long, and transection of the left rectus muscle at the level of the umbilicus. Localized peritonitis was present. The remaining peritoneal cavity showed no evidence of injury. The bowel was closed in two layers and the mesentery was closed with silk.

Postoperatively the course was uneventful Gastric suction was used, together with penicillin, streptomycin and intravenous Achromycin. He was discharged on the 6th postoperative day. Follow-up examination 3¼ years later showed no symptoms and no abnormal findings.

Case 2. S. J., an 8-year-old boy, was injured at 12 noon on June 23, 1957. While riding a bicycle, which suddenly stopped, his abdomen was thrust into the handle bar area, hitting his left lower quadrant. He was examined a few minutes later in his family physician's office. Because of the lack of findings he was sent home. He developed further abdominal pain and vomiting, and was admitted to the hospital 12 hours later with minimal abdominal findings. The following morning the abdominal findings were still insignificant. X-rays taken later that day showed free air in the peritoneal cavity. Surgical consultation was obtained 30 hours after injury, at which time there were signs of peritoneal irritation. The patient was observed for another 12 hours while he was being hydrated, and during this time the peritoneal signs progressed. Laboratory work was negative except for white blood count of 12,900.

Approximately 45 hours after injury a laparotomy was performed through a left paramedian incision. A rupture in the jejunum 25 cm. below the ligament of Treitz was found. It was 3 cm. in length and transverse in direction. It was well walled off by adjacent bowel and omentum. There was localized peritonitis below the left colon. The general peritoneum was amazingly clear. No other evidences of injury were found. A two-layer closure was done.

TABLE 1

Mortality from nonpenetrating small bowel rupture

Year	Author	Per Cent
1887	Curtis[2]	100
1902	Gage[2]	80
1935	Counseller and McCormick[3]	73.4
1942	Poer and Woliver[14]	61
1944	Ficarra[6]	56
1948	Bosworth[1]	29
1960	Martin *et al.*[9]	2.8

The postoperative course went well, with gastric suction, intravenous fluids, penicillin and streptomycin. The patient was discharged on the 8th postoperative day. Check-up 2½ years later was entirely negative, with normal growth and activity.

Case 3. I. T., a 55-year-old carpenter, was injured at 9 p.m. on September 10, 1957. He was sitting on a horse that reared on its hind legs and rolled over backwards. The saddle caught the midportion of the patient, the cantle hitting his abdominal region and the pommel hitting the right lower chest. The patient was hospitalized shortly afterwards although his general condition was good. Surgical consultation was obtained because of a possible chest injury. The abdomen was negative except for a bruise to the skin in the left midabdominal wall area. Chest x-ray was negative and the abdominal x-ray showed an unusual air pocket in the left upper quadrant of the abdomen (figs. 1 and 2). The following morning an obvious peritonitis was present and a diagnosis of ruptured jejunum was made before laparotomy.

A ruptured jejunum was found 24 cm. from the ligament of Treitz with a rent 5 cm. in length and localized peritonitis. There was no other evidence of injury.

Postoperatively the patient did well with gastric suction, intravenous fluids, penicillin and streptomycin. He was discharged on the 8th postoperative day. Follow-up 2½ years later showed no ill effects from the abdominal injury.

Case 4. M. S., a healthy 14-year-old boy, was playing baseball and was injured at 2:10 p.m. on April 14, 1959. As the patient was sliding into home base on his back, the catcher, who was a heavier boy, fell on him, his knee landing on the patient's midabdomen. The patient was unconscious for a few minutes. He had severe pain immediately, and within 2 hours had pain in his left shoulder and generalized abdominal pain. After emergency examination in the family phy-

TABLE 2

Nonpenetrating rupture of jejunum

Case No., Sex, and Age (in Years)	Time Interval until Operation	X-ray Findings	Location of Rent*	Discharge Day	Follow-up
	hr.				
1. M, 23	18	Negative	10 cm. + mesentary rent	5	3¼ yr.
2. M, 8	45	Free air under diaphragm	25 cm.	8	2½ yr.
3. M, 55	13	Air pocket under left colon	35 cm.	8	2½ yr.
4. M, 14	7	Negative	24 cm.	6	11 mo.
5. F, 38	28	No air; localized ileus	12 cm.	7	3 mo.

* Distances given indicate the distance from ligament of Treitz.

sician's office he was sent to the hospital. He had eaten lunch ½ hour before injury. In the hospital, 3 hours after injury, there were no bowel sounds; marked abdominal rigidity and tenderness were noted. Abdominal tap was negative, and x-ray showed no evidence of free air in the peritoneal cavity.

Approximately 7 hours after injury a laparotomy was performed through a right rectus incision. A perforated jejunum, 24 cm. from the ligament of Treitz, was found with a 2-cm. rent on the antimesenteric border. There was no other evidence of injury.

Postoperatively the course went well, with supportive care comprising gastric suction, intravenous fluids, and broad spectrum antibiotics. The patient was discharged on the 6th postoperative day. Follow-up 11 months later showed no residual effects.

Case 5. I. J., a 38-year-old, married housewife, was in an automobile accident at 7:20 p.m., December 15, 1959. She received a steering wheel type injury and was seen in the hospital emergency room 1½ hours later with chest pain. Although there was some midabdominal pain and low back pain, x-rays of the abdomen and chest were negative, with a suggestion of a soft tissue mass in the left midabdomen. This concurred with the physical findings of a subcutaneous hematoma on the left midabdominal wall anteriorly. The patient was moderately obese but had no other physical findings. She was admitted to the hospital for observation. Over the next few hours in the hospital the abdomen became more tender and bowel sounds disappeared. Rectal examination showed tenderness on the left. Peritoneal tap revealed a minimal amount of blood-tinged, clear fluid. Twelve hours later the abdominal findings were more pronounced, and a laparotomy was decided upon. The blood bank was unable to cross blood for this patient (blood type O-negative); therefore, surgery was delayed approximately 12 more hours, and 1000 cc. of blood were give preoperatively. Except for the low hematocrit, laboratory work was normal, with a mild elevation of the white count and a normal serum amylase. Repeat x-rays 12 hours after admission showed no free air, although there was some degree of localized small bowel ileus in the left upper quadrant.

A laparotomy was performed through a transverse incision in the left upper quadrant 28 hours after the injury. A perforation of the jejunum, 12 cm. from the ligament of Treitz, was present,

FIG. 2. Case 3, erect position

FIG. 1. Case 3, supine position

with a small rent on the antimesenteric border and localized peritonitis.

Postoperatively the patient did well. The gastric tube was removed on the 5th day and the patient was discharged on the 7th postoperative day.

DISCUSSION

All of the above cases are remarkably similar in their behavior and course. They correspond to other reported cases in the literature in which there has been an isolated injury to the upper jejunum. Two of the patients had the initial symptoms focused on a chest injury that proved to be insignificant. None of them had head trauma or other major trauma to mask the attention to the abdomen. All were hospitalized and observed adequately; all patients received a blunt blow to the midabdomen, probably left sided; all cases happened within 2 hours after the patient had ingested food. It can be speculated that the jejunum was relatively a "solid" viscus at this interval, allowing it to be compressed against the vertebral column or permitting a bursting effect from within to occur. The pathology was remarkably similar, a single rent, averaging 2 cm. in length, occurring on the antimesenteric border of the upper jejunum. Each patient had localized peritonitis limited to the left upper quadrant below the transverse mesocolon and behind the greater omentum. This localized protected process, together with the minimal spillage, accounts for the delayed diagnosis of peritonitis and surgical intervention. It has been speculated that a localized segment of bowel, near and above the rent, goes into spasm and thus prevents any leakage. Later the bowel relaxes, with peristalsis forcing intestinal contents out through the opening.[4, 5]

X-rays, flat and upright or lateral ducubitus of the abdomen, for the most part, did not help in the diagnosis. One patient showed free air under the diaphragm (20 per cent). One patient showed an unusual air pocket under the transverse mesocolon to correlate with the operative findings. None of the patients was drained.

In every case antibiotics were given postoperatively. We suspect this was not necessary. With the jejunal rupture at this high level there should be no more bacterial soiling than one would expect from a gastrojejunostomy. There should be much less chemical irritation than seen in a perforated peptic ulcer. With the current problem of resistant staphylococci, we believe we would manage a future case without antibiotics.

It is of interest that the laparotomy was delayed in case 5 because of the inability to cross-match blood for the patient. The surgeon decided to go ahead anyway, and after a few more hours blood was obtained. This brings up the question of whether the modern generation of surgeons are more apt to proceed with an early laparotomy because of the availability of safe anesthesia, blood transfusions and antibiotics. No doubt, if surgeons of 20 or more years ago were presented with our modern conveniences many of the patients making up the previous high mortality rates would have been saved with earlier laparotomies.

SUMMARY

1. Five cases of rupture of the jejunum due to nonpenetrating trauma and without significant other injury are presented. One case was diagnosed preoperatively.

2. The pathology in each was very similar, with a small antimesenteric rent in the upper jejunum, a localized peritonitis and a time lag in diagnosis and treatment.

3. All patients had ingested food within 2 hours of injury.

4. There was no postoperative morbidity and no mortality, which contrasts sharply with early reports.

5. Early laparotomy should be performed if this entity is suspected.

6. X-rays showed free air beneath the diaphragm in 1 case (20 per cent). One case showed an unusual picture with a free air pocket trapped under the transverse mesocolon. If recognized, this is specific for the diagnosis of this traumatic entity and should warrant laparotomy.

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Editorial

IMPROPER ATTIRE OF SURGEONS: A MEANS OF CROSS CONTAMINATION*

The careless and improper dress of surgeons, which is not infrequently observed in visits to hospitals and in illustrations for medical articles and advertisements, must be considered as one of the causes of hospital-acquired infections. Hospitals purchase expensive sterilizing equipment and disposable goods as a means of combating cross contamination; however, the importance of the proper operating dress of surgeons has received relatively little attention. Intimately related to this subject is the importance of having the finger-nails short, the scalp hair adequately trimmed and the skin free of pyogenic infection.

At one time it was generally considered that microorganisms, once gravitated to the floor, remained there and ceased to be a significant source of danger. More recently the floors and walls are being increasingly recognized as depositories of pathogenic organisms which contaminate the air of the operating room.[1] The organisms are transmitted by personnel on their clothes, hair and skin and in their respiratory excretions, as well as by such articles as bedclothes and stretchers.[2] As the surgeon, more than most other personnel, comes into contact with infectious cases on the wards and in his office, he should take particular precautions not to serve as a means of contamination to the operating room suite. Proper attention to his attire will reduce to a minimum the transmission of organ-

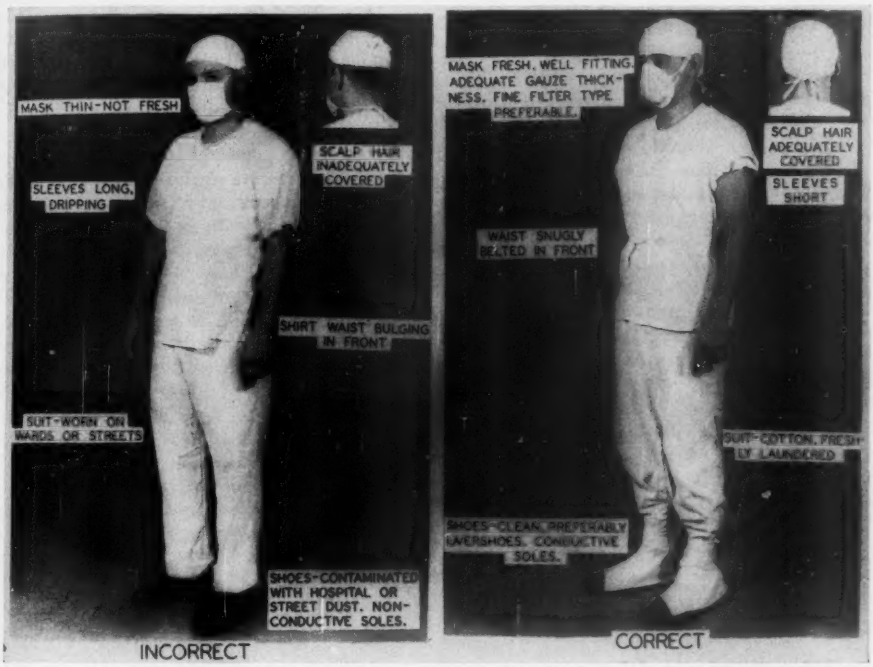
MASK THIN-NOT FRESH
SCALP HAIR INADEQUATELY COVERED
SLEEVES LONG, DRIPPING
SHORT WAIST BULGING IN FRONT
SUIT-WORN ON WARDS OR STREETS
SHOES-CONTAMINATED WITH HOSPITAL OR STREET DUST. NON-CONDUCTIVE SOLES.
INCORRECT
MASK FRESH, WELL FITTING. ADEQUATE GAUZE THICKNESS. FINE FILTER TYPE PREFERABLE.
SCALP HAIR ADEQUATELY COVERED
SLEEVES SHORT
WAIST SNUGLY BELTED IN FRONT
SUIT-COTTON, FRESHLY LAUNDERED
SHOES-CLEAN, PREFERABLY OVERSHOES. CONDUCTIVE SOLES.
CORRECT

FIG. 1

*From the Roper Hospital and Medical College of South Carolina, Charleston, South Carolina.

isms to and from the operating room by dust from his hair, clothes and shoes, and by his skin and respiratory excreta.

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BOOK REVIEW

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

The Transplantation of Tissues and Organs. By MICHAEL F. A. WOODRUFF, M.D., M.S. 777 pp. Charles C Thomas, Springfield, Ill., 1960.

This copious volume will surely be the classic reference work for years to come in the rapidly growing field of tissue transplantation. The author is himself an important investigator with many contributions of his own. The subject matter falls into two natural divisions. In the first 11 chapters the purely biologic aspects of the general subject of tissue and organ transplantations are dealt with in a most comprehensive and stimulating manner. The volume of quotations from the literature is very great, and the analysis of the various problems that present themselves is penetrating and judicious. This part of the work presumably was written largely for research workers in this field of biologic investigation, but most clinicians will find it of interest and broad appeal. The impression left is that of progressive accomplishment, and although much remains to be done, the specific attainments already recorded, the technical methods developed, and the identification of further concrete problems make it likely that the rate of progress will be even more rapid in the future.

The 12th to the 28th chapters, which comprise the remainder of the text, deal with the application of transplantations to clinical problems. Chapter 12 is given over to general clinical considerations. The succeeding chapters discuss the details of grafting methods. Following this, grafting of special tissues and grafting in particular anatomic regions receive extensive treatment.

The book is provided with a most extensive bibliographic index and also a separate authors' index and subject index.

The volume as a whole is a very impressive and valuable accomplishment. The one feature that may seem open to question is the rather frequent repetition of facts and opinions, but this is more or less unavoidable, because of the approach to the presentation of the subject. Thus the same situation may be dealt with as a part of the general biologic approach, as a part of the behavior of a certain type of tissue, and as a part of the problem characteristic of a certain anatomic area or structure. For a work of reference this repetition is unavoidable since the seeker of specific information may wish to find the answer to a question under any of the three general headings, principles, specific methods, or special anatomic areas, without reading the whole book through. But for those with the time and the interest to read the book as a whole, these repetitions do not seriously disturb the attention.

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